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THE SYNDROME OF PEUTZ

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Peutz[24] first described the syndrome of familial gastro-intestinal polyposis, associated with mucocutaneous pigmentation, in 1921. However, it was not until its 'rediscovery', and masterly account of 10 patients suffering from the condition, by Jeghers *et al.*[18] that it received wider recognition. Since then, well over 100 cases have been recorded in the literature, but the syndrome is still poorly recognized and an estimate of prevalence is thus impossible. It has been suggested that it is probably at least as common as familial polyposis of the colon.[12] This report includes 2 members of a family affected with the syndrome.

CASE HISTORY 1

1st Admission

Mr. E., aged 29 years, was admitted to the Johannesburg General Hospital on 18 April 1947 with intestinal obstruction.

He gave a 4-year history of episodic, colicky, central abdominal pain accompanied by nausea and vomiting. Such attacks lasted for about an hour, otherwise he was perfectly well. There was an associated weight loss of 30 lb. over the preceding 3 years. The present episode began a week before admission, with frequent colicky spasms lasting about 5 minutes and with severe vomiting. He had passed no faeces or flatus for 2 days before admission.

On examination, he was a thin sallow European male, with kyphoscoliosis. The abdomen was distended and visible peristalsis was noted. No masses could be palpated. Rectal examination was negative. Straight X-ray examination revealed several fluid levels, and large-bowel obstruction was diagnosed. No comment was made about pigmentation.

At operation, an ileo-ileal and caecal intussusception was found, with a mass the size of an egg in the ileal lumen. The draining lymph glands were enlarged. Twelve inches of small gut containing the tumour were resected and an end-to-end anastomosis performed. The patient made an uneventful recovery.

The tumour was fleshy and irregular, and seedling growths were present at its base. The pathologist reported that the mucosal tissue exhibited considerable irregularity with some evidence of penetration of the underlying muscle, which at one point had extended almost to the serosa. The neoplasm had the histological features of a highly differentiated adenoma showing changes 'suggestive of early malignant transformation'.

2nd Admission

The patient did not return to follow-up clinic after the operation, but was re-admitted on 29 September 1953 with a history of 3 years of recurrent attacks of abdominal colic culminating in the present severe episode.

On examination (A.J.L.) he presented with signs and symptoms of a small-bowel obstruction with a definite mass in the right iliac fossa.

In addition, typical pigmentation was noted on the face, particularly around the eyes and mouth and also in the mucous membrane of the upper lip. The Peutz syndrome was considered as a diagnosis.

At laparotomy, a small-bowel volvulus at the ileocaecal junction was present with an intussusception containing 4 ileal polyps (Fig. 1). About 3 feet of small bowel were resected with adjoining enlarged mesenteric glands. End-to-end anastomosis was performed. Further definitive exploration was not undertaken.

Histologically, the tumours were typical adenomatous polypi with no evidence of malignancy.

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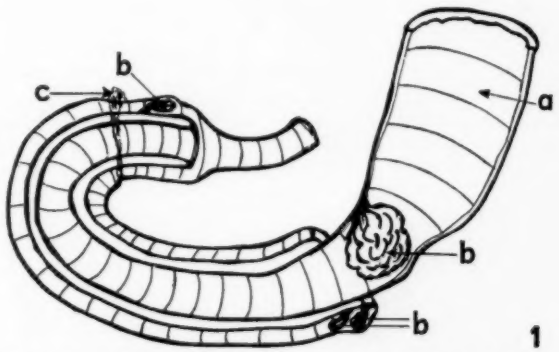

Fig. 1. Diagram showing findings at second operation (case 1)—a volvulus with intussusception containing 4 polypi. Note there is no polyp at the apex of the intussusceptum. (a= hypertrophied proximal bowel, b=1 large and 3 small polypi in the intussusception, c=fibrosis in previous anastomosis.)

3rd Admission

The patient was re-admitted on 15 February 1960 and one of us (I.F.A.) diagnosed sub-acute intestinal obstruction. The history was, as previously, of relief for about 2 years after operation, followed by the onset of recurrent abdominal pain. The pain was colicky and was now located in the right iliac fossa where a swelling had been noticed during attacks. This pain came on typically only during activity and was accompanied by marked borborygmi; it usually remitted suddenly and spontaneously, with the passing of flatus. The colic had been associated with diarrhoea and vomiting. There was no blood in the stool. On examination there was a ventral hernia in relation to the first operation scar. No masses were palpable. Dorsal kyphoscoliosis was noted. The haemoglobin was 13 G. per 100 ml. and occult blood was present in the stool. Barium studies could not be performed at the time.

Fig. 2. Typical cutaneous pigmentation on eyelids and around the orbits—case 1.

Operation was performed by Prof. D. J. du Plessis. The stomach appeared free of polypi on palpation. The small bowel was systematically examined and was hypertrophied throughout its entire length. Commencing at the proximal jejunum and working distally, a total of 5 enterotomies were performed and 15 pedunculated polyps removed. Wherever the bowel was opened the mucosa had a cobble-stone appearance from numerous commencing pin-head polypi.

The mesenteric glands were not enlarged. Other abdominal viscera, including large bowel, were normal.

The patient made an uneventful recovery. Histologically the tumours were adenomatous polypi of normal small-bowel epithelium with no evidence of malignancy. A double-contrast barium enema failed to reveal any evidence of colonic polyposis.

Typical pigmentation was noted around the eyes and on the eyelids (Fig. 2). However, the pigmentation of the lips previously noted was absent on this admission, and there was no involvement of the buccal mucous membrane. A random biopsy of the mucous membrane of the inner aspect of the upper lip was taken and showed a significant increase of melanin in the epidermis and dermis (Fig. 3).

Fig. 3. Section of mucous membrane from upper lip showing increase in melanin in dermis and epidermis (silver stain for melanin)—case 1.

CASE HISTORY 2

The only child of the above patient, a daughter aged 5 years and 10 months, presented at the Transvaal Memorial Hospital for Children on 4 June 1956 with a history of intermittent rectal prolapse for 3 years. The mother had replaced this after stool, and laxatives had been used to prevent undue straining. Two years previously the family doctor had noted rectal polypi and cauterized these. During the 3-year period of rectal prolapse she had suffered from cramp-like abdominal pain and had passed red blood in the stool on a few occasions.

At the hospital barium-enema studies showed no convincing evidence of polyposis coli. At operation a grape-like mass of polypi was removed from the rectum and 2 smaller ones cauterized higher up. No note was made at the time of pigmentation, but the parents recall that she had had 'marks on her lips since she was a baby'.

We saw this child recently, now aged 10 years, and she displays typical pigmentation—around the eyes and mouth, and most strikingly on the lips and buccal mucous membranes (Fig. 4). has been having frequent episodes of abdominal colic and 9

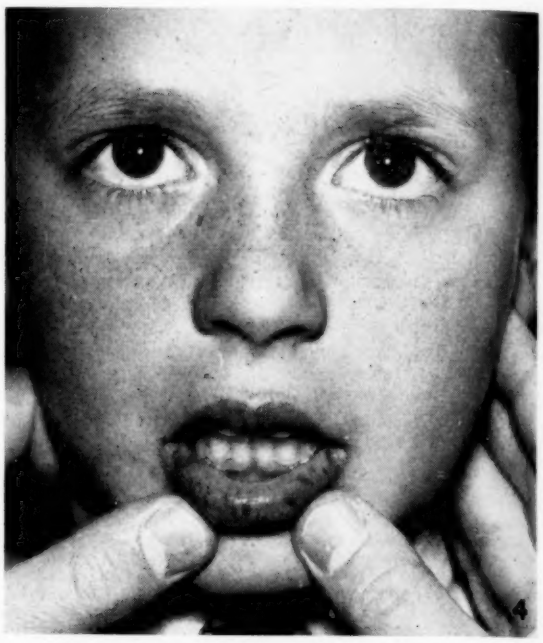

Fig. 4. Pigmentation of lips and peri-orbital region—case 2.

months ago had her appendix removed. We were unable to discover whether the appendix was acutely inflamed or not, but the persistence of cramps favours a diagnosis of intestinal polypi. Further examination of the child was not permitted.

DISCUSSION

The syndrome of Peutz has 3 cornerstones—heredity, polyposis, and mucocutaneous pigmentation.

Heredity

The polypi and the various forms of pigmentation are transmitted as Mendelian dominants[18] with a high degree of expressivity for both pigmentation and polyposis. The condition is heralded by pigmentation at birth or in early infancy. There is no particular racial or sex incidence.[3,28,30] The co-existence of 2 such apparently unrelated entities as polypi and pigmentation may be explained in several ways. Genetic linkage—the close association of 2 mutant genes—is untenable, because the 2 elements are seen apart too rarely for this. Pleomorphism is no longer a favoured explanation, but it is probable that the peccant gene has only a single action biochemically, many stages removed from the recognized clinical features.[23]

Various familial or congenital abnormalities have been reported in association with gastro-intestinal polyposis. Such an association may be purely coincidental or related to a general lowering of the phenotypic expression. A few patients with the Peutz syndrome have exhibited, in addition to the gastro-intestinal polypi, nasal and bladder polypi, and 1 patient had bronchial adenomatosis.[10,16,24] In view of the concept of 'visceral polyposis',[24] it is possible that the affected gene produces a disturbance at a critical stage of mucous-membrane development. Scoliosis has been noted by Tanner[23] and Dormandy[10] and was present in our adult patient. An

association with clubbing has been frequently noted,[9,18,20,26] while multiple skeletal malformations with soft-tissue tumours,[7] multiple bony tumours,[8] exostoses,[10] congenital heart disease,[10,18] diverticulitis and diverticulosis of the colon,[10] lateness of maturity,[18,20] and thyroid, adrenal and ovarian disease[6,8,18,33,35] have been reported less constantly. Pancreatic adenomatosis (Zollinger-Ellison syndrome) has been found in association with either pituitary, parathyroid or adrenal disturbance,[38] but has not been noted with intestinal adenomatosis.

Five genetic varieties of intestinal polyposis have been listed:[27] (1) Classical familial polyposis coli; (2) Peutz-Jeghers syndrome; (3) the Gardner syndrome (colonic polyposis associated with osseous and soft-tissue tumours); (4) the possible syndrome of Turcot, Deprés, St. Pierre (colonic polyposis and brain tumour); and (5) possible single isolated familial polypi.

Polyposis

The polypi are benign adenomata which may be sessile or pedunculated. They always arise in the small bowel, but may be found in the stomach or colon. They have not been described in the oesophagus. These polypi seem to erupt in different parts of the gut at different ages, with normal segments of gut intervening. Some polypi, notably those in the rectum, may undergo complete regression.[1] The majority of cases present with symptoms of gastro-intestinal polypi between infancy and middle age. Five of Dormandy's patients presented, several years before diagnosis of the full-blown syndrome, with rectal polypi in infancy, and this was well shown in our second case. Apart from the larger polypi, which can be removed, and the hundreds of pinhead growths which are seen at operation, Dormandy has stressed the existence of multiple intramural micro-adenomata in apparently normal-looking bowel. The common cause of obstruction is small-bowel intussusception, but the intussusception usually does not contain a polyp at its apex. The mechanism is probably local increase in peristalsis caused by micro-adenomata.

Histologically, the polypi give evidence of their origin from these intramural micro-adenomata by containing in their stroma interspersed thin and thick branching bands of muscularis mucosae. These bands are often dispersed among the glandular structures. The stroma may show infiltration with lymph, plasma and eosinophil cells. The normal glandular pattern of the small bowel is reproduced in the polypi (Fig. 5). This histological picture with the peculiar formation of the stroma and the reproduction of normal mucosal structure with multiple cell types has led to the belief that these polyps are developmental disturbances and are in fact hamartomas.[3,4,10,15,29] This same histological appearance may be interpreted as invasion of the muscular layer by the tumour and was probably responsible for many of the earlier diagnoses of malignant change. Our first case was diagnosed as showing an early malignancy of the small bowel, but review of the slide showed a typical adenomatous polyp. Several authorities agree that small-bowel polyposis in the Peutz syndrome is not precancerous.[10-12,32] There are two cases on record with undisputed carcinoma arising from a polyp.[2,5] Metastases have never been found arising from a polyp, nor have there been any reported deaths from carcinoma in the small bowel.[29]

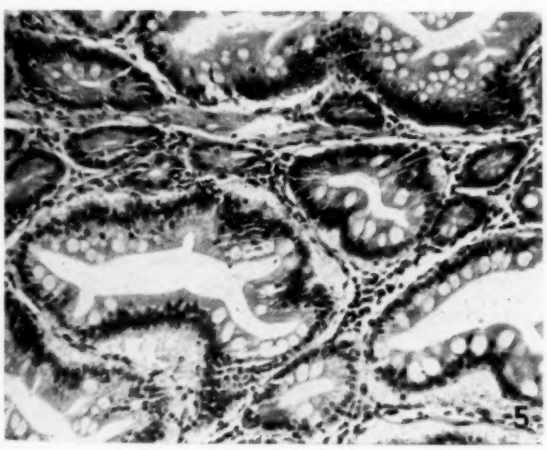

Fig. 5. Section of ileal polyp showing a normal glandular pattern with normal cells. Muscularis mucosae is seen branching between the glandular structures.

In contrast to the above, polypi in this syndrome, occurring in the colon and probably the stomach, are prone to undergo malignant transformation as often as any other polyp in this situation. A leading article in the *Lancet* put the matter nicely: There is something peculiar about the resistance of the human small bowel to malignancy. Its mucosa is extensive and it has a high mitotic rate. It lies in direct continuity with mucosae of the pylorus, bile ducts, pancreatic ducts, and colon—all areas of high cancer incidence.[14] Dormandy has commented on the apparent frequency of carcinoma of the rectum and colon in cases with the syndrome of Peutz.[13] We stress that, although the small-bowel polypi apparently do not tend to become malignant, those polypi in the colon and rectum must be regarded with great suspicion and be treated accordingly. It may be that a single pluripotential genetic error underlies both the Peutz syndrome and familial polyposis coli. Local factors in the colon, such as the end-products of digestion, may account for malignant change in the large-bowel polypi in both syndromes.

Pigmentation

The pigment is melanin and is probably present from birth. Cutaneous pigmentation occurs mainly around the facial orifices—mouth, nose, and eyes—and consists of small discrete round-to-oval macules not exceeding 3-5 mm. in diameter, with no tendency to coalesce. The colour varies from brown to black. Pigmentation has also been noted on the fingers, palms and toes, and more widely dispersed over the rest of the body.[25,33,40] The facial pigmentation usually fades during adolescence and may disappear altogether.[24] These spots must be differentiated from ordinary freckles, lentigo, chloasma, xeroderma pigmentosa, *cafe au lait* spots, and exogenous melanoses.

Histologically, the pigment has been described as occurring in vertical bands,[18] but most workers found no characteristic distribution.[10,31,34] Circumoral pigmentation without polyposis is not rare,[10,18,30,36] and familial polyposis without pigmentation has been noted.[17,39] Buccal mucosal pigmentation has been considered the *sine qua non* of the syndrome.[10,18] This was noted at first in our adult patient, but was strikingly

absent on his most recent admission, although a random mucous-membrane biopsy did show a significant increase in melanin (Fig. 3). The spots are about 5 mm. in diameter and are found on the lips, the buccal mucous membrane, hard and soft palate and nasal mucosa,[9] and more rarely the labial, conjunctival,[18] and rectal mucosae.[24] There is no pigmentation of the polyps, either in the small bowel or the colon.

Originally the syndrome was noted only in dark-haired, dark-complexioned patients,[18,33] but it does occur in fair-haired, light-complexioned people, although the pigmentation is then less obvious.[19,22,28] This is in accord with another family seen by one of us (A.J.L.) where the mother and 2 daughters with the Peutz syndrome were all fair-skinned.[21] Cases have not been reported in the Bantu.

DIAGNOSIS

Despite a family history and the presence of the characteristic pigmented spots, the condition is often misdiagnosed, as occurred initially in both our cases.

The patient usually presents with signs or symptoms of one or more of the following: (1) Intestinal obstruction; (2) bleeding and/or iron-deficiency anaemia; (3) intestinal hurry and malabsorption features; and (4) rarely with pigmentation.

Most commonly the patient is seen with enteric intussusception and obstruction. The clinical picture of the episodic colic, outlined previously, is so typical that a diagnosis can often be made on this alone. Overt rectal bleeding may be noted, but usually occult haemorrhage results in an iron-deficiency anaemia. Our young patient came to hospital with rectal prolapse. The increased peristalsis associated with polyposis may result in deficient absorption and stigmata of malnutrition. Rarely, the patients are seen at a dermatology outpatient department with a complaint of freckled 'dirty lips'.

Sigmoidoscopy and gastroscopy have limited diagnostic value and should be combined with double-contrast barium examinations of small bowel and colon.

TREATMENT AND PROGNOSIS

Surgical intervention may be life-saving but is never curative. Ultimately, cures will be achieved by correction of the underlying genetic biochemical disturbance.

With regard to the small bowel, treatment should be limited to symptomatic polyps and extensive resections are not advised.[10,12] In those cases with recurrent attacks, much depends on the frequency and severity of the episodes—if mild, simple measures may suffice. On the other hand, elective surgery may bring years of relief with minimal operative risk. Operation is carried out after adequate bowel preparation. A small incision is made on the antimesenteric aspect of the ileum about 25 cm. distal to the ligament of Treitz. Long Allis' forceps are inserted through this opening, and grip a full thickness of bowel wall as far away from the incision as possible. The forceps are then withdrawn and the bowel is intussuscepted through the incision. The mucosal surface may then be inspected under direct vision. About 20 cm. of gut on each side of the incision can thus be cleared of polyps—a total of about 10 enterotomies permits visualization of the whole small-bowel length.[37]

In polypi of the stomach, colon and rectum, malignant change is a distinct danger. Prophylactic surgery is indicated here, but 'cancer resections' are not justified prophylactically. Regular follow-up of these patients is imperative.

Overall life expectancy is not markedly reduced, and with careful supervision a reasonable prognosis is assured.

SUMMARY

A father and daughter affected by the syndrome of Peutz are presented.

The 3 main features of gastro-intestinal polyposis, mucocutaneous pigmentation, and heredity are discussed. The diagnosis is still too often missed and a wider recognition of the syndrome is called for.

The disappearance in later life of the typical mucosal pigment in the one patient is noteworthy.

The question of malignant transformation in the polyps is considered. The danger of such change occurring in the gastric or large-bowel area is stressed, and the necessity for prophylactic surgery and regular follow-up in these cases is emphasized.

Diagnosis and treatment are briefly alluded to.

We thank Prof. D. J. du Plessis and Prof. B. J. P. Becker for assistance; Mr. D. Smith and Mr. A. M. Shevitz for the photographs; and Dr. K. F. Mills, Superintendent of the Johannesburg Hospital, for permission to publish.

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VAN DIE REDAKSIE : EDITORIAL

DIE MEDIESE KONGRES IN KAAPSTAD

Die gereelde tweejaarlikse algemene mediese kongres van die Mediese Vereniging van Suid-Afrika word vanjaar gedurende die week 24 - 30 September in Kaapstad gehou. Die kongres word in die vroeë lente gehou, wat een van die Kaap se mooiste en gemoedelikste seisoene is. Die kongresprogram bied ook 'n wye verskeidenheid van aantreklikhede aan. Daar sal 'n kongresbal wees, 'n banket, 'n burgemeesterlike onthaal, geleentheid vir deelname aan allerhande sportsoorte, uitstappies, en spesiale onthale vir die vrouens en ander familielede van dokters. Ook sal daar 'n uitgebreide wetenskaplike uitstalling wees, 'n vertoning van stokperdjies, 'n program van beeldradio-uitsendings, en 'n omvattende uitstalling van farmaseutiese produkte en mediese instrumente en apparaat.

Die kern-waarde van die kongres lê egter in die uitstekende wetenskaplike program wat voorberei is. Om mee te begin sal daar twee voltallige sittings gehou word oor probleme wat op die huidige tydstip van die allergrootste belang is, nl. *Die versorging van bejaardes* en *Diabetes*.

Die Versorging van Bejaardes

Die probleem van die versorging van bejaardes is 'n onderwerp wat elke dag in belangrikheid toeneem. Soos ons al voorheen aangetoon het, is daar dwarsoor die wêreld 'n neiging tot relatiewe en absolute vermeerdering van die aantal bejaardes in die bevolking, en dit lei tot die ontstaan van mediese en maatskaplike probleme op hierdie gebied op 'n skaal wat vroeër heeltemal ondenkbaar was.

Die bespreking oor hierdie onderwerp sal oor 'n wye verskeidenheid van aspekte van die saak gaan, en sal by die kongres ingelei word deur mev. Zerilda Steyn, voorsitter van die Suid-Afrikaanse Nasionale Raad vir Bejaardes. Sy sal praat oor die *Allesomvattende versorging van bejaardes*. Dr. J. H. Sheldon, van Engeland, sal die probleem van *Oumense en hul gesondheid* bespreek. Dr. I. M. Hurwitz, van Kaapstad, se referaat handel oor *Die voorkoms van siekte of ongeskiktheid van persone wat in 'n tehuis vir bejaardes woon*, en dr. R. Getz, van Johannesburg, sal ook oor 'n tehuis van (Joodse) bejaardes praat. Mnr. S. P. Watson, van die Hulpvereniging van die Witwatersrand, sal die probleem van *Rehabilitasie van oumense* behandel. Dr. R. L. Retief, Direkteur van Hospitaaldienste in die Kaapprovinsie, sal praat oor *Die plek van die Provinsiale Administrasie by die versorging van bejaardes*, en die adjunk-minister van Volkswelsyn en Pensioene, adv. B. J. Vorster, sal die kongres toespreek oor *Die plek van Volkswelsyn in die versorging van bejaardes*.

Diabetes

Die vordering wat daar gemaak is op die gebied van navorsing oor die behandeling van diabetes gedurende die afgelope aantal jare, is niks minder as fenomenaal nie. Die studie van diabetes het eintlik 'n hele wetenskap op sy eie geword.

Ook hierdie onderwerp sal oor 'n breë front benader word. Die eerste deel van die bespreking oor diabetes sal handel oor *Diabetes in Afrika* en sal ingelei word deur dr. W. P. U. Jackson, van Kaapstad, wat internasionale bekendheid verwerf het deur sy werk op hierdie gebied. Daarna sal dr. G. D. Campbell, van Durban, praat oor *Onlangse waarnemings oor diabetes by Zoeloes en Natalse Indiërs*. Dr. H. C. Seftel, van Johannesburg, sal praat oor *Diabetes by Naturelle soos in Johannesburg gesien*. Dr. W. M. Politzer, ook van Johannesburg, sal die probleem behandel van *Diabetes by stedelike en plattelandse Naturelle*, en prof. J. A. Tulloch, van Kampala, sal praat oor *Diabetes — 'n vergelyking met ander dele van die Trope*.

Na die teepouse sal die probleem van middels teen diabetes wat per mond geneem kan word, bespreek word. Dr. D. Jackson, van Engeland, sal praat oor die *Chemie, farmakologie, en aanduiding vir die gebruik van middels teen diabetes wat per mond geneem word*. Drs. T. Schneider, van Johannesburg, en P. E. S. Loubser, van Bellville, sal altwee praat oor *Kliniese ondervinding met hipoglisemiese middels wat per mond geneem word*. Dr. J. B. Herman, van Kaapstad, sal die probleem van *Toleransietoetse vir glukose, en hipoglisemiese middels* behandel. In die namiddag sal daar 'n gekombineerde groepbespreking oor allerlei ander aspekte van die probleem van diabetes wees.

Om die kongres verder aantreklik te maak vir so 'n groot aantal dokters as moontlik, en om die eenheid van probleemstelling en gebied te beklemtoon wat tog nog bestaan ten spyte van die neiging tot spesialisasie en ‚super'-spesialisasie, het die kongreskomitee twee groot gekombineerde groepsvergaderings gereël, wat bygewoon kan word deur spesialiste uit verskillende vakgebiede en deur algemene praktisyns. By die eerste van hierdie groepsbyeenkomste sal die probleem van *Die diagnose en behandeling van afsluitende bloedvatsiekte* behandel word, en by die tweede die probleem van *Antibiotika, chemoterapie en kruisinfeksie*. Hierbenewens sal twee simposiums gereël word oor *Pleurale en pulmonale neoplasie*, en *Rumatiese hartsiekte*.

Behalwe die voltallige sittings, groepsbyeenkomste en simposiums, sal daar seksionele vergaderings wees in die verskillende vertakkings van die medisyne, en die Nasionale Groepe van die Mediese Vereniging sal geleentheid kry om hul besigheidsvergaderings te hou.

Prof. J. F. Brock, van Kaapstad, sal die openingsrede voer.

Langs hierdie weg wil ons weer 'n beroep doen op al ons kollegas om te doen wat hulle kan om van hierdie kongres 'n maatskaplike en wetenskaplike sukses te maak. Daar is reeds al by te veel geleenthede, en nie sonder 'n groot mate van waarheid nie, gesê dat die Mediese Vereniging te veel tyd spandeer aan ekonomiese sake in sy beraadslagings. Hier is 'n geleentheid om by te dra tot die sukses van 'n onderneming wat uit die aard van die saak 'n wetenskaplike en kulturele onderneming is. Laat ons die geleentheid om deel te neem aan produktiewe en skeppende optrede van hierdie aard nie misloop nie.

THE JUXTAGLOMERULAR CELLS

The juxtaglomerular cells are found mainly in the walls of the afferent arterioles in the kidneys near their junction with the glomerular network. They appear to be secretory cells and contain secretory granules. The granularity of these cells undergoes marked changes in various types of experimental hypertension and with variations in sodium intake.[1] Hypertension induced by experimental procedures tends to decrease the granularity of these particular cells. As the blood pressure through the kidney diminishes, the granularity tends to increase. An increase in the perfusion pressure through a normal kidney for 1½ - 2 hours will also produce marked degranulation. It is suggested that these cells act as stretch receptors, and change their rate of secretion as changes occur in the stretch of the walls of the afferent arterioles.[2,3]

The juxtaglomerular cells are also influenced by electrolytes. In laboratory animals a diet low in sodium significantly increases their granulation. Since this occurs not only in normal rats, but also in hypophysectomized rats, the pituitary gland does not appear to be important in this response. There is not so much certainty about the effect of a high intake of sodium. One explanation for the effects of dietary sodium on the granularity of the juxtaglomerular cells may be that a low sodium intake causes a lower extracellular fluid volume and blood volume, with presumably a lower volume in the afferent arterioles, and therefore diminished stretch of the cells. A diet with a high salt content may produce the opposite effects.

The results of various observations seem to indicate that the chief stimulus for increasing secretion by the juxtaglomerular cells is a decreased stretch of the cells resulting from low pressure in the renal artery or from a low volume of blood in the afferent arteriole. A decreased secretion is related to an increased stretch of the juxtaglomerular cells caused by increased pressure or increased volume of blood in the afferent arteriole. Very likely the secretory activity of the cells can change considerably without great changes in the number of visible granules. Only when the secretory rate is high or low will gross changes in the number of granules be seen. In other words, the granularity of the cells is only a rough indicator of their rate of secretion.

The juxtaglomerular cells and renin seem to be closely related.[1] It was suggested many years ago by Goormaghtigh[4] that the granules contain renin. Definite evidence has been obtained in recent years that the main supply of renin is in areas containing glomeruli and not in purely tubular areas of the kidney. It has, in fact, been shown that renin is located in or near the glomeruli, most likely in the juxtaglomerular area. This does not rule out the possibility that juxtaglomerular cell granules contain some other substance in addition to renin or its precursor. Experiments have shown that renin is very likely part of some regulatory system and not merely a structural protein in the kidney. Its relationship to the granularity of the juxtaglomerular cells supports this view.

A great deal of work has been done to establish a relationship between the juxtaglomerular cell-regulating system and hypertension. There is good evidence that a change in arterial pressure locally in the kidney arterioles will change the rate of secretion of these cells. It may be that the cells secrete renin in response to a diminished stretch of the arteriolar walls. It would appear that renin as a direct pressor agent does not maintain the hypertension, but the kidney may have some antihypertensive action. It may be a function of the kidney to secrete an antihypertensive hormone. When an insufficient amount of this hormone is being secreted, the smooth muscle cells in the arterioles take up more sodium, potassium, and water, swell slightly, and increase their tone. More information is needed before a true understanding of the rôle of the juxtaglomerular cells and changes in their activity can be related to essential hypertension in man. The literature is vast, and detailed information must be read elsewhere.[1] The whole subject is obviously of more than academic interest.

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THE SOUTH AFRICAN MEDICAL CONGRESS, 24 - 30 SEPTEMBER 1961, CAPE TOWN

HOTEL ACCOMMODATION

The Forty-third Medical Congress of the Medical Association of South Africa will be held in *Cape Town* from 24 to 30 September 1961. Information regarding the Congress is published at regular intervals in the *Journal*. Members who intend coming to Congress are requested to send in their Intention Forms as soon as possible.

Prospective visitors to the Congress are also requested to bear in mind that *hotel accommodation in Cape Town is somewhat limited* and that they should make the necessary reservations well in advance of Congress. The Travel Bureau of the South African Railways has been appointed the official agent in this connection. Members are requested to contact their local agents who have been extensively circularized in this respect. In the small platteland towns the nearest stationmaster will handle the matter.

Make your reservations now in order to avoid disappointment. In the event of any difficulty, please write to Dr. J. C. Coetzee, Convener, Accommodation Sub-Committee, 43rd South African Medical Congress, P.O. Box 643, Cape Town.

TRANSPLANTATION OF URETERS FOR VESICO-VAGINAL FISTULA

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There is some difference of opinion about the usefulness and safety of uretero-colic anastomosis for the treatment of vesico-vaginal fistula. Mahfouz[1] maintained that very few patients live more than 3 years after such an operation. Most authorities, however, are agreed that the outlook for those treated for vesico-vaginal fistula and other non-malignant conditions is comparatively favourable.

In some instances the operation is nothing less than life-saving—for instance in a patient shown by Belloso[2] at La Sociedad de Cirugia del Uruguay, in whom endoscopic resection of the neck of the bladder, performed for retention of hysterical origin, had caused a fistula. Three attempts at repair per vaginam failed. The urethra had become a rigid tube, and the patient, after 2 years of chronic invalidism, was driven to the verge of suicide. Transplantation of the ureters was then carried out, with the result that within 17 days there was great general improvement, disappearance of the hypochondria, and well-controlled micturition. Satisfactory progress was maintained.

I have noticed an extraordinary improvement in the physical and mental state of African patients following this operation. They had all been admitted with the vagina in an extremely septic condition, and none of them regretted having undergone the operation; from being outcasts among their people they have been rendered socially acceptable.

Arthur Jacobs,[3] in a survey of 1,673 uretero-colic anastomoses, stated that the postoperative mortality for traumatic conditions, the majority of which were vesico-vaginal fistulae, is the lowest for any of the classified conditions for which the ureters were transplanted into the colon. The expectation of life after the operation for vesico-vaginal fistula is second only to that for congenital anomalies.

These findings are in accordance with my experience in Nyasaland, where women whose ureters were transplanted several years previously by different surgeons have returned to hospital pregnant or seeking treatment for some other complaint. In one patient, upon whom a hysterectomy had to be performed for rupture of the uterus caused by obstructed labour, great anxiety was occasioned and operating time was wasted because the ureters could not be found in their normal position; they had been transplanted into the upper part of the sigmoid colon some years previously. The patient had stated that the abdominal scar found on examination was from a Caesarean section, and she had apparently forgotten the operation which she had undergone for transplantation of her ureters.

Another patient (case 5 described below) had been operated upon for transplantation of her ureters in 1954. She returned in 1960 complaining of incontinence, having continued to pass urine per vaginam for the intervening 6 years. At operation 2 more ureters were found which had been overlooked at the first operation.

THE OPERATION

Indications

Transplantation of the ureters from the bladder to the colon or upper rectum is indicated when a vesico-vaginal fistula is so extensive that local repair is impossible. It should also be considered when attempts at closure of the fistula would be unlikely to succeed on account of thickening and rigidity of the walls of the fistula, vaginal stenosis, incontinence of the sphincter urethrae membranaceae, or urethral stenosis, and when attempts at suture have failed.

The question arises of how many attempts should be made to eliminate a vesico-vaginal fistula of moderate dimensions before resorting to uretero-colic implantation. Some women in Nyasaland have been subjected to several such attempts, and success has finally been achieved.

McCord,[4] in his fascinating book *My Patients were Zulus*, described a Zulu woman upon whom he operated 7 times in attempts to repair a vesico-vaginal fistula. 'The seventh attempt at last sealed up the vaginal outlet, converting bladder and vagina into one sac—not the ideal solution, but at least affording her fair control over her discharges so that she would no longer be offensive in the kraal.' On being informed that his wife would be unable to bear more children, the patient's husband declared that he did not wish to have anything more to do with her!

Contraindications

1. The presence of an irreparable recto-vaginal fistula.
2. Pronounced loss of tone or incontinence of the anal sphincter. This can be rectified by a preliminary operation, but sufficient time must be allowed for the wound to become soundly healed before transplanting the ureters.

Pre-operative Investigation

Laboratory facilities here are limited and there is no radiologist on the hospital staff. Examination is performed to determine:

1. The size of the vaginal outlet and the extent of infection;
2. the presence or absence of urethral stenosis;
3. the size and position of the fistula, and whether it involves the urethra;
4. the condition of the sphincter ani;
5. whether a recto-vaginal fistula is present;
6. the blood-urea level;
7. the condition of the kidneys and the number and size of the ureters by intravenous pyelography; and
8. by examination of the stool, whether intestinal bilharzia, hookworm, or other infection is present.

Permission is obtained, if possible, for performing salpingectomy in those likely to be endangered by further pregnancies. Most Nyasa women refuse such permission, for childbirth is considered by them and their husbands to be the most important reason for their existence, whatever its sufferings and dangers.

Pre-operative Treatment

Vaginal infection is treated for several days. The bowel is rendered as clean as possible by the administration of appropriate sulphonamides or antibiotics, and a urinary antiseptic is prescribed if the intravenous pyelogram shows evidence of pyelonephritis or ureteric thickening. A purgative is given 48 hours and an enema 24 hours before the morning of the operation. A low-residue diet is then prescribed and 1 or 2 colonic washouts are given on the day preceding the operation.

Technique

Coffey's technique[5] has been fairly closely followed in 4

cases; other methods were used when for any reason Coffey's technique could not be employed. Simplicity, with reduction of operating time and therefore of shock, has been aimed at. The anaesthetic used has been open ether.

With the patient in Trendelenburg's position, and with an intravenous infusion instituted, a median subumbilical incision is made. The intestines are packed off and a self-retaining retractor is inserted. The right ureter is found and traced downwards as far as the broad ligament. It is divided there and the distal end is ligated. The left ureter is dealt with similarly. The right ureter is then dissected up towards the brim of the pelvis, until sufficient length is obtained for the anastomosis. The arteries seen to be supplying it are preserved, taking some of the peritoneum with the ureter if necessary. The lower end of the ureter is slit longitudinally for half an inch, and the short extremity transfixed distally with 00 chromic catgut with a straight needle at either end. The ureter is tied on both sides so that it is firmly grasped by the stitch. The end is wrapped in gauze and laid aside. The peritoneum is brought together in the place where the ureter lay.

The left ureter is dissected up similarly. When the length available is too short to allow it to be implanted below the pelvic mesocolon, it is first carefully freed below, then drawn upwards to a position above the mesocolon. This is done by thrusting the ends of a pair of long artery forceps through a small incision in the parietal peritoneum of the posterior abdominal wall above the mesocolon. The points of the forceps are passed downwards retroperitoneally, as suggested by Jidigan and Bickers,[6] behind the root of the mesentery, care being taken to avoid damage to the inferior mesenteric vessels and their branches. The forceps grasp the end of the ureter, drawing it upwards so that it emerges above the mesocolon, where it is split distally, transfixed with a catgut stitch, secured on either side, and wrapped in gauze.

The ureters are now implanted into the colon through 2 separate incisions. On the right side the incision is made for about ¾ inch obliquely downwards and forwards through the peritoneal and muscular coats as far as the anterior taenia. The mucosa is defined, separated by blunt dissection, pulled upwards, and incised transversely at the lower end of the incision. The 2 straight needles carrying the ends of the catgut stitch fastened to the ureter are introduced separately into the lumen of the bowel through the small mucosal incision, and directed downwards and medially to a point 1 inch below the incision and about ¼ inch apart, where they are made to emerge through the bowel wall. Traction on these ends pulls the tip of the ureter neatly through the mucosal incision into the bowel. The ends are firmly tied, and the ureter is securely anchored. Failure to introduce the needles accurately low enough down the lumen of the bowel will allow urine to leak backwards into the peritoneal cavity.

Interrupted catgut stitches are used to close the seromuscular incision, all but the lowest being made to pick up the wall of the ureter superficially. The anastomosis is covered, if possible, with a flap of peritoneum.

The left ureter is implanted into the bowel similarly. If this is done above the pelvic mesocolon, the pelvic colon is rotated over to the right to receive the ureter without strain and anchored by a catgut stitch, passed from an appendix epiploica to the psoas muscle or other convenient anchorage.

A wide rubber tube with side fenestrations is then passed by the right hand into the rectum from below, the left hand being used in the abdomen to guide it up the bowel to the level of the upper anastomosis, as recommended by Hickey.[7] In my experience this is a very important step, since free drainage of urine *via* a rectal tube greatly reduces the danger of leakage of urine into the peritoneal cavity. After removing gloves and recleansing the hands, the rectal tube is secured with nylon to the skin close to the anal sphincter. Rubber dam drains are introduced through the abdominal wound down to the anastomoses, and the abdomen is closed.

The advantages of implanting the left ureter low down into the recto-sigmoid rather than higher up above the mesocolon are that the path taken by the ureter is straighter, that it is easier afterwards to pass a rectal tube for drainage of urine right up to the level of the anastomosis, and that there is no need to immobilize the pelvic colon in an abnormal position. Should a sigmoidoscope be passed subsequently by a surgeon unaware of the fact that the direction of the pelvic colon has been altered, the instrument might possibly damage the colon or cause a drag on the ureter.

Postoperative Treatment

1. Intravenous fluids are administered until the patient is fit to take water freely by mouth, and gastric suction is instituted if necessary.

2. Water and glucose only are allowed until flatus has been passed, then a liquid diet is given until the rectal tube has been removed.

3. Aneurine hydrochloride, 250 mg. daily, is given subcutaneously for 4 days or until faeces and flatus are passed through the rectal tube.

4. An intestinal antiseptic, such as phthalylsulphathiazole, is continued for 1 week, and a urinary antiseptic is given as well if there is any likelihood of urinary infection.

5. The rubber dam abdominal drains are removed on the fourth day.

6. On the seventh day the rectal tube is removed, a mild purgative is administered if necessary, and the patient is allowed to get up.

CASE REPORTS

Case 1

Elizabeth, aged about 20 years, was admitted to hospital on 2 November 1959 with a vesico-vaginal fistula after the birth of a stillborn child following her first pregnancy. The vagina was contracted and there was a hard fibrous band on the posterior wall. First degree utero-vaginal prolapse was present. The urethra was impermeable. The pelvis was grossly contracted, but salpingectomy was refused.

Transplantation of the ureters was performed by Coffey's method on 17 November, but the ends of the ureters were pulled down only ½ an inch into the bowel, and the sutures burying the ureters were introduced rather loosely for fear of constricting the lumen. A rectal catheter was passed, but it was not brought up to the level of the anastomosis. Two days later, much urine was leaking from the abdominal wound as well as through the rectal tube. The condition of the patient deteriorated, probably from peritoneal irritation following imperfect drainage. A second operation was therefore performed, and the anastomoses examined. The right was satisfactory, but urine was exuding through a small hole in the region of the left. Omentum was stitched over the leak, and a large drainage tube introduced. Three days later, paralytic ileus developed.

The flow of urine through the abdominal wound gradually ceased and all urine was passed per rectum. On 9 January 1960 an intravenous pyelogram showed that both ureters were functioning. The blood-urea level was satisfactory, and the patient had good control over both urine and faeces. She was discharged feeling well, with the wound healed.

Case 2

Nekelece, aged about 30 years, was admitted to hospital on 11 November 1959 with a fistula 1 × 1½ inches in size. The vaginal walls were extremely indurated and there was almost complete absence of the perineum. The anal sphincter, however, still retained some tone, and there was no incontinence of faeces. The usual preliminary treatment was given, including a course of stibophen for intestinal bilharziasis.

The ureters were transplanted on 15 December. Both were enlarged to the thickness of a little finger. They were divided and dissected upwards for about 2 inches, the intention being to transplant them by the method of Brendan Hickey,[7] a modification of that of A. Davalos, in which a transverse incision is made across the anterior surface of the colon through the sero-muscular layers, and a half-moon of these layers dissected downwards, leaving the mucous membrane intact, which is then divided at the lower end of the half-moon. The mucous membrane was, however, intimately attached to the outer layers. Attempts to dissect down the half-moon caused buttonholing of the mucosa in 2 places. Long-standing bilharzial infection of the bowel had evidently caused adhesions of the mucous membrane to the muscular coat.

The ureters were therefore pulled down the colon with stitches for 1 inch, and the bowel closed round them as tightly as possible, securing the thickened outer coats of the ureters to the bowel wall. A large rubber tube was passed per rectum up to the level of the anastomoses, through which urine started to drain by the time the operation was completed.

An intravenous pyelogram on 6 January 1960 showed that both ureters were excreting well, and the patient was discharged symptomless the next day. She has returned twice for re-examination, when her condition was entirely satisfactory except that leakage of urine occurred from the rectum while she was asleep, probably on account of lack of the normal tone of the anal sphincter.

Case 3

Chembotele, aged about 28 years, was admitted to hospital on 12 December 1959 with a large fistula which had developed after the birth of a stillborn child following her first pregnancy 4 years previously. A band of scar tissue surrounded and narrowed the vaginal outlet. The anal sphincter was satisfactory. An intravenous pyelogram showed poor excretion of the right kidney, and the appearances suggested a double left ureter. After the usual preliminary treatment, the ureters were transplanted by Coffey's method on 5 January 1960. The pyelographic findings were misleading, for there proved to be only one ureter on the left side. The postoperative course was uncomplicated, and good control over micturition was established. The patient absconded from hospital on the 11th postoperative day.

Case 4

Ellen, aged about 27 years, was admittted to hospital on 27 January 1960 with incontinence of urine since November 1959, when she had given birth to a stillborn child, having been in labour for 3 days after her only pregnancy. Examination revealed stenosis of the vaginal outlet and a large communication between the bladder and the vagina. Intravenous pyelography showed good excretion down the left ureter in ½ an hour and down the right in 1 hour. The ureters were transplanted by Coffey's method on 9 February. The convalescence was uncomplicated, and an intravenous pyelogram showed satisfactory excretion down both ureters 30 minutes after injection of the medium. The patient was discharged on 3 March.

Case 5

Grace, aged about 30 years, was admitted to hospital on 28 February 1960 with a history of incontinence since 1954 after the birth of a stillborn child, having been in labour for 4 days. The ureters were stated to have been transplanted 2 months after this, and a record of the operation was found in the register, but her incontinence had continued ever since.

A large fistula was found, admitting 2 fingers easily, with prolapse of the bladder wall into the vagina. A catheter introduced into the urethra was held up by an impassable stricture ½ an inch from the meatus. The rectal sphincter was normal.

An intravenous pyelogram showed 4 ureters, but the shadows cast by the medium were not very clear. The blood urea was 60 mg. per 100 ml. The stool contained hookworms.

Operation revealed 1 ureter on the left and a very small ureter on the right still entering the bladder. Another ureter on the right side had been transplanted, and a structure which was thought to be a ureter on the left side was entering the pelvic colon. The untransplanted ureter on the left was divided as low down as possible, but it was not sufficiently long to reach the rectum. It was therefore inserted into the pelvic colon above the mesocolon. The minute extra ureter entering the bladder on the right was divided between ligatures, since it was too small to transplant. Recovery was satisfactory. Good control of micturition was established and there was no further leakage from the vagina.

Case 6

Mphangela, aged about 25 years, was admitted to hospital on 11 May 1960 with a history of incontinence for 1 year following childbirth. A fistula was present, for which an attempt at closure through an abdominal incision had been made at another hospital 4 months previously.

A catheter could be made to enter the bladder easily *via* the urethra. The fistula, about 4 × 3 inches, replaced almost the whole of the anterior wall of the vagina. The anal sphincter was fair. The vulva was excoriated by an eczematous condition. An intravenous pyelogram showed good excretion from the right kidney at 35 minutes; fair from the left kidney.

The ureters were transplanted by Coffey's method on 17 May. Progress was satisfactory until 1 June, when a small quantity of urine was observed leaking through the abdominal wound, and an intravenous pyelogram showed some leakage from the left ureter, but by 21 June the wound had healed. Thd patient was discharged free of symptoms.

Case 7

Mulimba, aged about 28 years, was admitted on 31 January 1960. Three weeks before admission she had given birth to a stillborn, full-term child. A large slough was seen partially closing the vagina, into which urine and faeces were discharging. A large fistula replaced most of the anterior wall of the vagina, and a circular recto-vaginal fistula, about 1¾ inches in diameter, could be felt 2¾ inches from the anus. The urethra was stenosed and the anal sphincter was lacking in tone. Two days later, the large foul-smelling slough was removed from the vagina.

It was hoped, after a preliminary colostomy, to repair the recto-vaginal fistula, and later to transplant the ureters into the pelvic colon. A right transverse colostomy was accordingly performed on 23 February. Later, the spur was divided, and the lower segment of the bowel irrigated.

Two attempts were made by a colleague, Dr Winifred Lochrie, to repair the recto-vaginal fistula, but healing was prevented by constant fouling of the wound with urine, even with the patient lying face downwards, for the vagina was greatly contracted—almost nonexistent in its upper part—and there was a profuse growth of granulation tissue. The anal sphincter was, however, successfully repaired at the first operation.

In view of the irritating effect of the constant flow of urine into the vagina, it was decided that further attempts to repair the rectum without first diverting the urinary stream would fail.

On 28 June, through a left lower rectus-split incision, a bilateral partial salpingectomy was performed, and an ileal ureterostomy effected by the method described by Pyrah and Raper.[8] The continuity of the ileum was restored in front of the isolated ileal segment by side-to-side anastomosis. Difficulty was experienced in obtaining a water-tight junction between the thickened ureters and the isolated loop of ileum. After the anastomosis had been completed, and the distal end of the ileum brought out in the right iliac fossa, it was found that the urine did not drain externally, but was escaping into the peritoneal cavity. It was therefore necessary to shorten the ileal loop and perform a second, tighter anastomosis of both ureters. This was done in much the same way as in performing a uretero-colic anastomosis.

A Foley's catheter was used to drain urine from the ileal loop, but it became blocked repeatedly with blood clot and mucus. Greater success was attained with a de Pezzer catheter, but this also did not drain well unless slight traction was applied to it. Later, an ordinary rubber tube was employed, being held in position by passing it through a hole in a piece of corrugated rubber strapped to the abdominal wall. Fig. 1 shows the ureters after transplantation and the drain from the ileal bladder.

Fig. 1. Intravenous pyelogram of case 7 to show the outlines of the ureters after transplantation into an isolated loop of ileum. This X-ray was taken 55 minutes after injection of the opaque medium. The drain from the ileal bladder is also seen.

Paralytic ileus occurred after the operation, but recovery followed. The patient is being fitted with a Birkbeck appliance. On the advice of Professor Pyrah, no further operations will be undertaken for several months. It is hoped to repair the rectum, implant the ileal bladder into the pelvic colon, and close the colostomy, but these steps are being postponed until all possible inflammatory reaction in the region of the recto-vaginal fistula has completely subsided. The patient is now in good health. The blood urea is 60 mg. per 100 ml., but she has no cerebral, gastro-intestinal, or respiratory symptoms, is free from headaches, and does not suffer from thirst.

COMMENT

Only one of the patients (case 2) whose ureters were transplanted has returned for follow-up observation, so that details of the subsequent progress of the other patients cannot be given. This is inevitable, for most of those treated came from places some distance away, and it is not usual for Africans to report once they think they are cured, unless complications develop. As far as is known there have been no deaths.

In conclusion, the following points are stressed:

1. The patient should receive sufficient fluids during the first few days after the operation. Parenteral administration of fluids may be necessary for more than 48 hours.

2. The passage of a rectal tube right up to the level of the anastomosis, if possible, greatly assists the free drainage of urine.

3. The possibility of the presence of more than 2 ureters must be borne in mind, even if these do not show on the intravenous pyelogram, since failure to transplant all the ureters will lead to a continuance of the passage of urine per vaginam.

SUMMARY

1. Some advantages of uretero-colic anastomosis in the treatment of vesico-vaginal fistula are quoted.

2. The indications and contraindications are summarized.

3. Suggestions are made for the pre-operative investigation and treatment, and postoperative care.

4. The technique of the operation, as practised by me, is described.

5. Reports are included on 7 consecutive patients. In one with a recto-vaginal fistula as well, an artificial bladder was constructed by isolating a segment of small intestine.

ADDENDUM

The report on case 7 (Mulimba) was not complete when this article was prepared. The following notes cover her progress up to April 1961. Mulimba was discharged in January 1961 wearing a Birkbeck appliance, which is a rubber bag connected to a shield secured with adhesive material to the abdominal wall. The urine flows into the bag, which is changed and washed out frequently. A colostomy bag and belt were worn in addition. The patient returned at intervals from January to April 1961 for inspection and any necessary renewal of parts of her equipment. When I last saw her, on my departure from Lilongwe in April, her condition was satisfactory and she had agreed to continue using the bags indefinitely. No further operation is contemplated.

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ISOLATION OF SPONDWENI VIRUS FROM FOUR SPECIES OF CULICINE MOSQUITOES AND A REPORT OF TWO LABORATORY INFECTIONS WITH THE VIRUS

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Spondweni virus was first isolated in 1955 from a pool of *Mansonia* (*Mansoniodes*) *uniformis* (Theo.) mosquitoes collected in northern Zululand, South Africa.[1] It was shown then that Spondweni virus belonged to Casals' group B of the arthropod-borne viruses, and a survey of human sera collected from residents of northern Zululand showed the presence of neutralizing antibodies against this virus.

Shortly after the isolation of this virus, a field station was established by this Unit in northern Zululand where periodic field investigations have been made in a continuation of our study of the arthropod-borne viruses. This field station is at Ndumu which is about 20 miles from the Simbu area where the original isolation of Spondweni virus was made.

During the period March — June 1958, intensive field investigations were carried out at Ndumu, since previous experience had shown that overt viral activity is at its peak at this time of the year, namely, the late summer and autumn.

From mosquitoes collected at Ndumu during this period 9 strains of Spondweni virus were isolated. While these strains were being handled in the laboratory, 2 cases of illness occurred among laboratory personnel. Evidence is presented which indicates that these illnesses were caused by Spondweni virus.

MATERIALS AND METHODS

The main investigations at Ndumu consisted of the following activities:

1. Collection of mosquitoes for virus isolation.
2. Collection of tissues from small mammals and birds for virus isolation.
3. Collection of blood specimens for virus isolation from persons with symptoms suggestive of a virus infection who attended clinics in the area.
4. Collection of blood specimens from small mammals and birds for antibody studies.
5. Use was made of human beings and animals as sentinels. Antibody studies on sera obtained from bleedings at the beginning and end of the observational period were made to investigate possible antibody conversions.

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The mosquitoes were caught off vegetation by hand in glass test-tubes, 90×15 mm., which were then plugged with cotton wool. At the field station the mosquitoes were grouped according to species and collecting sites. They were then stored on CO_2 ice until processed in the main laboratory in Johannesburg for inoculation into mice. For this the mosquito lots were processed in 0·75% bovine plasma albumin and the suspension from each lot was inoculated intracerebrally (IC) into 2 litters of infant mice, 2 - 3 days old, and into 6 adult mice. The dose of inoculum was 0·03 ml. for mice of both ages. Further details on the techniques used for the collection and handling of mosquitoes for virus isolation are given elsewhere.[2,3]

During the period March — June 1958, almost daily mosquito collections were made at 12 collecting sites. Since the establishment of the field station at Ndumu, many different collecting sites had been tried and the ones exploited during 1958 were those which had yielded the greatest number of mosquitoes. A collecting site is a small area usually about 50 yards in diameter. It is a clump of the somewhat denser scrub or trees and is relatively well shaded. Some of them are not in the immediate vicinity of water.

The serological procedures used in the neutralization (N), complement-fixation (CF), and haemagglutination-inhibition (HI) tests follow the methods described by Smithburn,[4] Casals *et al.*,[5] and Clarke and Casals,[6] respectively.

For use in the N test, the antiserum against the Spondweni prototype strain (AR 94) was obtained from a vervet monkey following 2 intraperitoneal inoculations of the virus. The AR 1038 antiserum was a pool from 2 rabbits following a single IC inoculation of virus. In CF and HI tests mouse hyperimmune antisera were used.

ISOLATION OF THE VIRUS STRAINS

Between 18 March and 5 June a total of 13,123 female mosquitoes was caught and inoculated into mice. Nine strains of virus, which were subsequently identified as Spondweni virus, were isolated from 4 species of mosquitoes as indicated in Table I. Included in this table are 3 mosquito species from which virus was not isolated, but which were obtained in relatively large numbers. The mosquitoes listed in this table comprise 98% of the total catch.

In mice these strains behaved similarly to each other and also to the original Spondweni virus strain, AR 94, isolated in 1955. By IC passage of infected mouse brain no difficulty

TABLE I. SPECIES OF MOSQUITOES COLLECTED AT NDUMU, MARCH-JUNE 1958, WHICH WERE PREVALENT AND/OR FROM WHICH STRAINS OF SPONDWENI VIRUS WERE ISOLATED

Species	*No. per lot*			*Total lots*	*Total mosq.*	*Spondweni virus isolations*	*Virally productive collection site*
	Min.	*Max.*	*Mean*				
Aedes (*Neomelaniconion*) *circumluteolus* (Theo.)	12	300	100	51	5,097	4	4, 16, 18, 21
Mansonia (*Mansoniodes*) *africana* (Theo.)	1	166	30	31	926	3	4, 18, 21
Aedes (*Aedimorphus*) *cumminsi* ssp. *mediopunctatus* (Theo.)	3	25	10	3	31	1	18
Eretmapodites silvestris (I. and de M.)	1	8	2	7	16	1	16
Aedes (*Aedimorphus*) *albocephalus* (Theo.)	2	228	88	52	4,563	0	—
Culex (*Culex*) *univittatus* (Theo.)	6	138	38	37	1,396	0	—
Mansonia (*Mansoniodes*) *uniformis* (Theo.)	3	173	33	25	835	0	—

was encountered in establishing the 9 isolates in mice. In the case of the first strain isolated, AR 1038, the infant mice were noticed sick or dead on the 5th and 6th days following inoculation of the mosquito suspension. The adult mice inoculated with the same suspension sickened between the 8th and 10th days. On IC passage of brain from a sick infant mouse into further mice, infant mice sickened on the 4th day and died on the 5th and 6th days, while in adult mice deaths occurred a day or two later. From the 2nd passage, AR 1038 virus, as well as the other 8 strains, were fully lethal for mice of both ages. All the strains passed readily through Seitz asbestos filters.

In four instances where a mosquito suspension had yielded virus, the original suspension was inoculated into mice after storage at about −20°C. in an attempt at re-isolation. Of these suspensions, 3 proved infective for mice.

IMMUNOLOGICAL IDENTIFICATION OF THE ISOLATES

HI. From all 9 isolates a haemagglutinin was obtained from an alkaline aqueous suspension of infant mouse brain after centrifugation at 10,000 r.p.m. for one hour. This suggested their relationship to the group B viruses. HI tests with various specific antisera confirmed this relationship and their probable identity as Spondweni virus. In these tests their group B properties resulted in inhibition by several group B antisera, but by no other antisera. The highest titres occurred with a Spondweni hyperimmune mouse serum. Other group B viral antisera which gave inhibition were notably West Nile and, to a lesser extent, Uganda S and Wesselsbron.

CF. The identification of all 9 isolates as Spondweni virus was established in CF tests with the same antigens used in the HI tests and a range of mouse hyperimmune viral antisera. These antisera consisted of antisera against 4 group A viruses, 5 group B viruses and Rift Valley fever and Simbu viruses. Fixation occurred with Spondweni antiserum and with none of the other antisera.

N. The identity of the isolates was confirmed with the N test. All these tests were carried out in adult mice inoculated IC. As indicated in Table II a Spondweni virus antiserum significantly neutralized the 9 isolates. In addition there was reciprocal cross-neutralization between one of these isolates and Spondweni virus.

TABLE II. RESULTS OF N TESTS BETWEEN AR 94 STRAIN OF SPONDWENI VIRUS, 9 STRAINS OF VIRUS ISOLATED FROM MOSQUITOES, AND AR 94 AND AR 1038 VIRUS ANTISERA

Virus	*Serum*	
	AR 94	*AR 1038*
AR 94	3·3*	3·8
AR 1038	2·4	2·9
AR 1071	2·8	
AR 1077	3·6	
AR 1081	3·0	
AR 1084	2·1	
AR 1086	2·1	
AR 1163	3·4	
AR 1168	2·5	
AR 1266	3·3	

* Results expressed as the neutralization index.

LABORATORY INFECTIONS

As mentioned previously, while work with the Spondweni isolates was in progress in the laboratory, 2 cases of illness occurred among laboratory personnel. The details of these cases are as follows:

Case 1

J.L., a White female, 21 years old, became ill on the evening of 23 June 1958. Her symptoms were generalized aches and pains, rigors, and vertigo. Her temperature at 8 a.m. on 24 June was 100°F., but was normal when a blood specimen was obtained at 1.30 p.m. on the same day. It was observed that her blood clotted very slowly. Recovery was quick and complete.

Case 2

C.B., a White male, 19 years old, became ill on 27 April 1958. His symptoms were severe headache, weakness, nausea, and slight epistaxis. On the morning of 28 April his temperature was 99°F. and in the afternoon 102·3°F., when a blood specimen was collected. On the 29th his temperature was 99·5°F. and a slight headache was present. The patient remained in bed until 1 May when he returned to work. However, he went off duty that afternoon owing to a severe headache and weakness and stayed away from work for a further 3 days.

Serum obtained from the acute-phase blood specimens of both individuals was inoculated into infant mice in an attempt at virus isolation. A virus was isolated from J.L.'s serum, but not from that of C.B. An attempt at re-isolation by inoculation into mice of J.L.'s serum after 14 days' storage at −20°C. was unsuccessful. However, this can possibly be explained by a loss of infectivity during storage, since its original infectivity was such that only 6 out of the 12 infant mice inoculated sickened. As is evident from the serological studies on this serum mentioned below, the group B virus antibody present in this serum may also have been a factor in the low infectivity.

The virus obtained from J.L. was identified as Spondweni virus in an N test. In this test a Spondweni virus (AR 94) antiserum neutralized 2·5 logs of the virus.

The antibody response following the illnesses of the 2 patients was studied by means of the N, CF and HI tests.

Owing to the antigenic overlap between the group B viruses, 5 other group B viruses, apart from Spondweni virus, were included in the CF and HI tests with sera from both individuals. Also included in these 2 tests was a pre-illness serum from both patients as well as a second convalescent serum from J.L.

The results obtained with sera from C.B. and J.L. in the CF and HI tests are shown in Tables III and IV.

TABLE III. CF RESULTS WITH SEVERAL GROUP B VIRUS ANTIGENS AND VARIOUS SERA FROM INDIVIDUALS J.L. AND C.B.

	Sera from J.L.				*Sera from C.B.*		
Antigen	*7 Feb. 1958 pre-illness*	*24 June 1958 acute*	*10 July 1958 1st conv.*	*10 Mar. 1959 2nd conv.*	*28 Mar. 1958 pre-illness*	*28 April 1958 acute*	*28 May. 1958 conv.*
Spondweni (AR 94)	0	0	0	4*	0	0	8
Wesselsbron ..	0	4	32	8	0	0	0
H 336† ..	0	32	128	64	0	0	0
West Nile ..	0	8	32	16	0	0	0
Zika	0	8	8	8	0	0	0
Yellow fever ..	0	16	128	16	0	0	0

* Reciprocal of highest serum dilution giving +3 fixation.
† A group B virus isolated from man in South Africa and related to, but not identical with, Uganda S virus.[11]

TABLE IV. HI RESULTS WITH SEVERAL GROUP B VIRUS ANTIGENS AND VARIOUS SERA FROM INDIVIDUALS J.L. AND C.B.

	Sera from J.L.				*Sera from C.B.*		
Antigen	*7 Feb. 1958 pre-illness*	*24 June 1958 acute*	*10 July 1958 1st conv.*	*10 Mar. 1959 2nd conv.*	*28 Mar. 1958 pre-illness*	*28 April 1958 acute*	*28 May 1958 conv.*
Spondweni (AR 94)	0	40*	640	320	0	640	1,280
H 336	0	1,280	2,560	1,280	0	0	40
Wesselsbron ..	0	320	640	320	0	0	40
Zika	0	10	40	10	0	0	0
West Nile ..	0	1,280	2,560	2,560	0	0	160
Yellow fever ..	10	160	320	160	0	0	0

* Titre expressed as reciprocal of highest serum dilution which gave inhibition.

From Table III it can be seen that Spondweni CF antibodies were present in the convalescent serum and absent in the acute and pre-illness sera. Also CF antibodies against the other 5 group B viruses were absent in the convalescent serum. In Table IV it can be seen that Spondweni virus HI antibodies were absent in the pre-illness serum, while there was a two-fold rise in antibodies following the illness. Of the group B viruses tested, the HI titre was highest against Spondweni virus.

In the N test C.B.'s acute serum gave no neutralization of Spondweni virus, whereas the convalescent serum was fully protective against 158 LD_{50} of virus.

In the case of J.L., preliminary tests indicated that the antibody response following her illness was by no means clear-cut. This individual had been immunized some 5 years previously against yellow fever and it appeared likely, from the HI and CF tests on her acute-phase serum, that she had had a group B virus infection between the time of commencement of her employment in this laboratory in February 1958, and her illness in June 1958. During this period she had been intensively engaged in the preparation of several group B virus HA antigens so that opportunity for such infection to have occurred had existed.

Four sera were available from J.L. for antibody tests. These included a pre-illness serum collected on 7 February 1958, when she started work in this laboratory; the acute serum, from which Spondweni virus was isolated, obtained on 24 June 1958, the day following the onset of the illness; and 2 convalescent sera. The one was obtained on 10 July 1958, 16 days after the onset of the illness, and the other on 10 March 1959. During January 1959 she had received a second yellow fever immunization. The results with the CF, HI and N tests on these sera are shown in Tables III, IV and V.

TABLE V. RESULTS OF N TESTS WITH SPONDWENI VIRUS, A NORMAL MONKEY SERUM, AND THE ACUTE AND CONVALESCENT SERA FROM PATIENT J.L.

Serum	*Logs virus titre*	*Logs virus neutralized*
Normal monkey	5·5	—
Acute, 24 June 1958	4·1	—
Convalescent, 10 July 1958 ..	2·8	1·3

With the CF test (Table III) it can be seen that, while her pre-illness serum was devoid of antibody against all the group B agents tested, her acute serum contained antibody against 5 of these agents, but not against Spondweni. It is noteworthy that after 16 days following the commencement of her illness Spondweni antibody was still absent, but there was an increase in antibody titre against 4 of the other agents. Also, whereas her second convalescent serum showed the appearance of Spondweni antibody, the antibody titres against the remaining group B agents either remained constant or decreased.

From Table IV it is evident that at the time of her illness the patient possessed quite a degree of group B virus HI antibody which reached a considerably higher level at the time her first convalescent serum was obtained. In this increase it should be noted that there was a sixteen-fold rise in antibody titre against Spondweni, while only a two-fold or four-fold rise occurred against the other antigens. The 1 : 10 HI titre against yellow fever antigen in the pre-illness serum is possibly the result of her first yellow fever immunization.

The results of the N test on J.L.'s acute and first convalescent sera shown in Table V, indicate a slight rise in Spondweni virus antibody during convalescence. While a neutralization index of only 1·3 was obtained, this figure was reproducible and this low value could possibly be accounted for by the presence of group B virus antibody in the acute-phase serum. The difference in titre of the virus in this serum and a normal monkey serum supports this contention.

DISCUSSION

The isolation of Spondweni virus from 9 different pools of mosquitoes caught at Ndumu was conclusive evidence of the presence of this agent in the immediate vicinity. These isolations occurred after more than 2 years of work at Ndumu during which Spondweni virus had not been isolated. The apparent sporadic appearance of viruses in Tongaland has been observed with other arthropod-borne viruses during the course of our studies in this area.

Of the 12 collecting sites at Ndumu which were exploited, 4 yielded virus. The productive sites are located within an area about 6 miles in diameter which was virtually the area of our field activities at Ndumu at this time. Within such narrow limits, these isolations must reflect an intense type of viral activity.

The non-arthropod hosts involved in this outbreak can only be speculated upon. Our observations suggest that birds and small mammals should be excluded. Attempts at virus isolation were made from 120 wild birds and 85 small mammals caught during the period March — June 1958, with no isolation of Spondweni virus. Although the birds were obtained just outside the perimeter of the mosquito-collecting area, the mammals were trapped at the actual collecting sites. Furthermore, antibody studies on sera obtained from birds and rodents have shown that Spondweni-neutralizing antibody is not prevalent in these animals from this area.

The susceptibility of cattle, goats and sheep, of which there are large numbers in the area, is not known. Of 234 sera collected in Tongaland in 1955 from cattle, sheep, and goats, tests for neutralizing antibody against Spondweni virus were positive in only 6 instances.[7] However, only 2 of these positive sera were monotypic for Spondweni virus alone, since the other sera were also positive against other group B viruses. In the area of the 1958 Spondweni outbreak, use was made of 31 cattle and 8 sheep as sentinels. Antibody studies with the N test on paired sera from these animals showed that 3 cattle and 2 sheep had possibly been infected with Spondweni virus during the outbreak.

What rôle the several thousand human beings resident in the Ndumu area may have played in the outbreak was not determined. Our attempts at virus isolation from 53 febrile patients who attended the clinics and from whom blood samples were taken, resulted in the isolation of a strain of West Nile virus,[8] but no Spondweni virus. N tests on paired sera from 7 African boys employed as mosquito catchers showed that one of these boys had an antibody conversion to Spondweni virus. On the other hand, the incidence of Spondweni antibody was found to be extremely low in 111 sera collected in December 1958 from Africans resident in the Ndumu area. These sera were initially tested in an HI test when 23 were positive at a dilution of 1 : 10 to Spondweni antigen. When these 23 sera were tested in an N test, only 2 were fully protective against Spondweni virus.

Aedes circumluteolus and *Mansonia africana* both frequent the shady bush habitat, although the latter species is also known to enter dwellings irregularly at night.[2] Visual observation[2] and the results of precipitin tests on recently engorged mosquitoes of these 2 species caught in the Ndumu area have shown that they possess somewhat similar host preferences, namely, they feed mainly on man and the larger domestic and wild animals, but infrequently on birds and rodents.[9]

Despite the host preferences of 2 species of mosquito implicated in this outbreak, our failure to isolate virus from the human beings who attended the clinics and the lack of more definite serological evidence incriminating man, cattle, and sheep means that the vertebrate host must remain conjectural.

Theiler and Casals[10] reported on the nature of the antibody response following infection with yellow fever in human beings, some of whom had apparently been infected previously with another group B virus. A distinction was made between the antibody response following a primary group B virus infection and that following a secondary infection. In a primary infection the response was more specific to the infecting agent, especially with the N test, and, if heterologous antibodies developed, they did so to a lower titre than the homologous antibodies. In a secondary infection the antibody response was to a variety of agents, and the only conclusion that could be derived from serological evidence was that a group B agent was involved. In these cases a specific diagnosis can be made only if the causal group B virus is isolated during the illness. Often the highest antibody levels following secondary infections were heterologous.

In accordance with these findings and the results of our serological tests on their sera, C.B.'s illness is considered a primary group B infection and J.L.'s illness a secondary or even multiple group B infection if the first yellow fever immunization is taken into account. Hence, the diagnosis of Spondweni infection in these cases would depend on the following evidence:

C.B.

1. The absence of Spondweni and group B, CF antibodies in the acute serum.
2. The presence of neutralizing Spondweni and CF antibody in the convalescent serum and the absence from this serum of CF antibody against 5 other group B agents.
3. The fact that HI antibody titre in the convalescent serum was highest against Spondweni virus.

L.J.

1. The isolation of Spondweni virus from the acute serum.
2. The rise in group B virus antibody during convalescence.

SUMMARY

1. Nine strains of Spondweni virus were isolated in mice from 13,123 female mosquitoes collected in Tongaland, South Africa, in 1958.

2. Four strains were isolated from *Aedes* (*Neomelaniconion*) *circumluteolus* (Theo.), 3 strains from *Mansonia* (*Mansoniodes*) *africana* (Theo.), and one strain each from *A. cumminsi* ssp. *mediopunctatus* (Theo.) and *Eretmapodites silvestris*, Ingram and De Meillon.

3. With the haemagglutination-inhibition, complement-fixation and neutralization tests, these strains were found to be immunologically similar to the strain of Spondweni virus isolated in Tongaland in 1955.

4. The outbreak of Spondweni virus activity in mosquitoes in 1958 followed a 2-year period when the presence of this virus had not been detected in Tongaland.

5. The non-arthropod host involved in the outbreak was not apparent.

6. At the time that strains of Spondweni virus were being handled in the laboratory, 2 cases of illness occurred among laboratory personnel. Spondweni virus was isolated from one individual. The nature of the antibody response in the convalescent sera of both was studied with the neutralization, complement-fixation, and haemagglutination-inhibition tests.

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BREAST ABSCESS

SELECTED CASE REPORTS

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Two recent articles[1,2] have drawn attention to the large number of breast abscesses seen at the Groote Schuur Hospital, Cape Town. Various aspects of the problem of treatment are discussed, including the probable routes of infection, the rôle of maternity homes, the methods of treatment, and the use of stilboestrol, antibiotics, and chemotherapeutic agents.

In this paper 6 cases are presented which serve to illustrate certain aspects of the infection:

CASE REPORTS

Case 1

R.C., Coloured female, aged 29 years. This patient developed a small painful mass in her left breast 3 weeks after the birth of her second child in a maternity institution. Her breasts were small and underdeveloped, lactation was poor, and the child had been bottle-fed after a few unsuccessful attempts at breast feeding. One week later she was referred to the hospital for treatment.

The abscess was incised and drained. Postoperatively there was brisk bleeding from the incision. The blood loss was suffi-

cient to warrant replacement by transfusion. She was discharged the following day feeling quite well. Two days later she was seen again and was now obviously very ill. She had had a rigor the previous day, and was now pyrexial and toxic, complaining of pain in the neck, back and buttocks, and had a persistent headache. On examination her temperature was 102°F. and she had minimal neck stiffness. There had been no further bleeding from the incision. No other significant finding was noted. She was admitted to hospital, blood was taken for culture, and furaltadone was given. Her temperature rose to 105·5°F. and after 48 hours chloramphenicol was administered as well. During this time she developed a mild transient glycosuria. Fasting blood sugar was normal. The fever subsided by lysis within 1 week, but she still had much pain in the lower back. This pain subsequently settled in the right sacro-iliac region. Repeated X-rays ultimately showed changes in the right sacro-iliac joint which suggested early infection. A diagnosis of pyogenic arthritis of the joint was made.

Blood culture, which was positive on the first occasion only, and pus from the breast both showed a *Staphylococcus aureus* with identical antibiotic responses. A similar organism was cultured from nasal and throat swabs from the patient's baby. He subsequently developed a bronchopneumonia from which he recovered.

The patient continued to have pain in the right sacro-iliac region, but this gradually diminished in severity. Furaltadone was continued until the erythrocyte sedimentation rate was normal on the 44th day. She was fitted with a corset and discharged. The breast incision had healed at the end of the second week in hospital. The pain in her back improved gradually until finally, after manipulation, her movements were normal and pain free.

This case illustrates a pyaemia resulting in the formation of a pyogenic arthritis following a lactational breast abscess. It is a rare complication—the only one in our series of more than 500 cases.

Case 2

D.P., Coloured female, aged 22 years. The patient presented with a 5-day history of pain and swelling in her left breast. Two weeks previously her first child had been born at home, with a nurse from a maternity institution in attendance. She had received 5 daily injections of penicillin and streptomycin, and had received parenteral and oral stilboestrol to suppress lactation.

A large abscess involving a major portion of the breast was incised and drained. Furaltadone was prescribed. Large masses of slough were discharged through the incisions, and after 3 days daily dilute eusol irrigations were employed. The haemoglobin evel was 10 G. per 100 ml. and iron was prescribed orally. On the 20th day she developed 2 small superficial abscesses, one on her left calf and the other in the right cubital fossa. It was decided that the original incisions were no longer providing adequate drainage, and re-incision with loop drains was accordingly carried out. Erythromycin was substituted for the furaltadone. Three weeks later healing was complete.

This case illustrates the extensive breast-tissue destruction resulting from delay in treatment, and also a transient pyaemic process which subsided without further complication.

Case 3

I.R., White female, aged 22 years. A week after the birth of her first child this patient developed a painful swollen left breast. While still in the maternity home she was treated with penicillin, streptomycin, and oxytetracycline. She was seen 5 days later in the Casualty Department, Groote Schuur Hospital, with a large left breast abscess. Her haemoglobin level was 10·5 G. per 100 ml. Oral iron was prescribed. The abscess was incised and drained. Five days later she still showed constitutional symptoms of the infection and erythromycin was prescribed. On the 9th day the left breast required re-incision and an abscess of the right breast was incised simultaneously. On the 29th day another abscess in the right breast was incised. After this, healing was uneventful and she was finally discharged on the 57th day.

This case illustrates recurrent abscess formation even though the patient was receiving the appropriate antibiotic. This occurs in about 10% of our cases.

Case 4

M.R., White female, aged 23 years. During her stay in the maternity home following the birth of her first child this patient developed a tender mass in her left breast. She received antibiotics and continued to breast feed her child; the abscess settled, leaving an indurated, non-fluctuant, and pain-free mass. Lactation was normal. Three months later she presented with a painful swelling of the right breast of 2 days' duration. The mass in the left breast was still present and showed no signs of inflammation. The abscess in the right breast was incised and drained, and furaltadone was prescribed. Two days later she required incision of the previously quiescent mass in the left breast. At this time her haemoglobin level was found to be 9·5 G. per 100 ml. She was given a blood transfusion and iron therapy was commenced. On a further 3 occasions incision and drainage of recurrent abscesses had to be carried out. At the last of these the previous incisions were re-incised and multiple adequate loop drains were inserted. After the 29th day novobiocin was substituted for furaltadone. Healing now progressed normally and she was discharged on the 47th day.

Culture of the pus from the breast abscesses and from nasal and throat swabs of her baby showed *Staphylococcus aureus* with identical antibiotic responses. The baby at no time showed any clinical evidence of harbouring the organism.

This case illustrates the formation of recurrent abscesses, notwithstanding the use of antibiotics and chemotherapeutic agents with demonstrated *in vitro* activity against the causative organism. It also illustrates the reactivation of an apparently resolved abscess.

Case 5

M.M., Coloured female, aged 28 years. This patient was first seen 3 weeks after institutional delivery of her first children, twins. Postpartum haemorrhage was excessive and necessitated blood transfusion. During convalescence in the nursing home she developed a painful inflammatory swelling in her left breast which subsided on antibiotic treatment. When she attended hospital her obvious anaemia and the size of the abscess of her left breast were striking. The haemoglobin level was 7·5 G. per 100 ml. This was an iron-deficiency anaemia for which oral iron was prescribed.

She was given a blood transfusion pre-operatively, and at operation 24 oz. of pus were drained, and loop drains were employed. Postoperatively a large portion of the breast parenchyma sloughed through the incisions. Daily dilute eusol irrigations in conjunction with systemic furaltadone were employed. Healing was retarded by the formation of a superficial abscess in the same breast. This was incised and drained, and chloramphenicol was substituted for the furaltadone. She was finally discharged on the 47th day. The breast was considerably smaller than the right. It was non-tender, slightly indurated, and slightly pigmented. The haematological picture was normal.

This case illustrates the extreme anaemia and the extensive tissue destruction that accompanies prolonged pus formation.

Case 6

F.B., Coloured female, aged 20 years. This patient was delivered early one morning in the Casualty Department, Groote Schuur Hospital. Her child weighed 3 lb. 10 oz. and was immediately transferred to the paediatric unit. Postpartum blood loss was negligible. The haemoglobin level was 10 G. per 100 ml., and oral iron was prescribed. A week later she presented with an abscess in her right breast of 2 days' duration. It was incised and drained and furaltadone was prescribed. Uneventful healing was complete by the 15th day and she was discharged.

The interesting features here are the following: The patient spent only about an hour and a half in the hospital. The child was never put to the breast. Lactation was minimal and required no suppression. The patient was anaemic. The response to early treatment was satisfactory.

COMMENT

One feature common to these cases is a greater or lesser degree of anaemia. Certainly the infection itself aggravates the anaemia, but in view of Lanskowsky's[3] findings, and the high incidence of breast abscesses seen in the non-European patients attending this hospital, anaemia may well have an aetiological significance.

In the great majority of patients seen at this hospital[1] and in all the cases reported here, the responsible organism is the ubiquitous *Staphylococcus aureus*. In the cases mentioned the organisms had similar antibiotic responses. They were insensitive to the commonly employed agents, viz. sulphatriad, penicillin, streptomycin and tetracycline. It appears that one of the reasons for delaying incision and drainage was that a trial of one or more of these antibiotics was being carried out. The use of antibiotics and chemotherapeutic agents in the treatment of breast abscess is not beyond dispute. Undoubtedly they have real value in treatment of the infection before abscess formation. Engelbrecht[1] showed that there was little to be gained from their use, although the use of furaltadone[2] produced encouraging results.

It must be stressed, though, that *Staphylococcus aureus* produces powerful exotoxins with potent tissue-destroying properties, and also that the coagulase secretion of this organism tends to reinforce the natural fibrin barrier produced by the host in an attempt to localize the infection.[4] The abscess thus produced in the resilient tissues of the breast is well protected from the action of systemic therapy. This is the strongest argument in favour of early definitive treatment of breast abscesses by incision and drainage.

SUMMARY

Six cases of breast abscess selected from a large series are presented to illustrate certain features of the disease. Attention is drawn to the anaemia accompanying the disease. The mode of abscess formation by *Staphylococcus aureus* is mentioned. Early incision of breast abscesses is advocated.

I wish to thank Dr. J. G. Burger, Superintendent of the Groote Schuur Hospital, for permission to publish these cases; Prof. J. H. Louw, Head of the Department of Surgery; and Mr. T. Schrire, Surgeon-in-charge of the Casualty Department, Groote Schuur Hospital.

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THE VALUE OF THE MEDICAL ASSOCIATION*

F. N. GILLWALD, *President, Orange Free State Goldfields Branch* (*M.A.S.A.*), 1960

The choice of a subject for a valedictory address is not always easy, but, luckily, recent events in this Branch have provided a ready-made theme.

This Branch is today officially one year old and has, I venture to say, conducted its affairs with efficiency and honour. The retiring Branch Council has set a high standard for its successors, and the incoming Council has a solid foundation on which to build. To conduct its affairs, heavy expenditure is incurred, and it became obvious at a very early stage that the most pressing internal problem would be that of finance. Office expenditure, delegates, expenses and official functions all absorb money, but they are vital functions of the Branch. Do not forget that your Branch has exactly the same commitments as the older Branches which have had years to accumulate such funds as they possess. They were able to do this, in most cases, before the present rapidly increasing tempo of medical politics.

Dr. Gillwald

At one of our Quarterly General Meetings the proposal was adopted that a levy of R20.00 should be placed, for this one year only, on each member of the Branch, to avoid a recurrence of the cramping financial restrictions of the past year and the necessity of asking for additional funds from time to time, especially on occasions such as this meeting. Now that the time has come when we must actually dig into our pockets, an outcry against the levy is heard from many quarters. In discussing the matter, one of the objectors asked, 'What do I get *out* of the Medical Association?' Now, I think (and hope) that what he meant was 'What extra do I get out of the Branch for this large amount of money you are asking?', because I find it difficult to understand why every medical practitioner of whatever category does not belong to the Association and actively support it. No sensible, thinking person can fail to see the advantages of having a nation-wide, unified body speaking for the whole profession and guarding its interests. In actual fact, non-members of the Association are being carried by the Association and receive many of its benefits, particularly in the financial field.

However, they deprive themselves of many of the other advantages that come with membership of the Association, and I would like to list a few of these advantages that come readily to mind. These include the right to attend clinical and other meetings organized by the Association, culminating in the biennial Medical Congress, where the best that South Africa has to offer is on show and is strongly reinforced by overseas visitors who are leaders in their respective fields; the value of the *South African Medical Journal* with its scientific articles written mainly by South African doctors, its instructive editorials, its information on current trends in medical politics, and its advertising facilities; the opportunities of joining cheap 'group' insurance plans, the Professional Provident Society and other schemes aimed at providing additional security for members at the cheapest possible rates.

Other advantages are: participating membership of the Medical Services Plan, which we all hope will be able to extend its field of action rapidly throughout the country in the near future; the right of appeal to the Association in disputes with Medical Aid Societies and other organizations, and the vast experience available in the settlement of such disputes represented by the full-time secretarial staff in Cape Town and Pretoria; the protection offered by the Branch Ethical Committees (with their confidential methods of operation) against attacks, whether justified or not, by patients or others; the social activities of the Association, either those after more serious meetings, or the purely social, which represent an important advantage to members, allowing them to relax with colleagues, to meet old acquaintances, make new friends, and bring their wives into communal medical life in recognition of the assistance they give on so many occasions; and finally there is the Benevolent Fund which is there to help in times of great necessity.

From the wider view of the Medical Association, let us focus our vision on the Orange Free State Goldfields Branch. Does it justify the R20.00 we are asking from you? I think that the answer will always remain a personal one. Those who take an active part in the work of the Branch, meeting the problems and solving them, or taking them through the various levels and Groups until a solution is reached by one of the higher Committees or Councils, will, I think, find the money well spent. Those who support the Branch, but less actively, will have to decide whether the higher status, in medical eyes, of being an independent Branch, is worth while in terms of money. The voices of our representatives are now heard directly in the highest Councils, without the delays our previous status involved. We can speak of our problems with the

*Valedictory address, 4 March 1961.

authority of local knowledge, and have wider powers to deal with these problems without reference to or permission from any outside body. Surely these advantages are worth something?

Let those who feel that they get nothing out of the Association come to us and discuss their problems, air their grievances and show us where we can improve. The Medical Association desires to be all things to all its members, but each individual member sees only his own small sphere, and it is only by getting together and working together that the many different viewpoints can be correlated, unified and turned to the best advantage of the profession through the Association. Those who criticize the Association on one or another aspect have probably much justification, for the Association is far from perfect; it has made some sad errors in the past and will do so in the future. But the Association is trying to live up to Article 3 (*a*) of the Memorandum of Association, which states that the object of the Association is 'to promote the medical and allied sciences and to maintain the honour and interests of the medical profession'. It can most nearly attain this ideal if every practitioner in the country supports it actively, criticizes it constructively, and gets to know and understand its many problems.

Those members of this Branch, who do not see their way clear to paying the levy, need not feel bad about it if their convictions are sincere. There is no call for drastic protests in the way of resignations and so on. The Branch can manage somehow without their money, but it would really hate to lose their moral support. Therefore I trust that those who do not pay will make every effort to attend the General Meetings and other meetings to lend their voice to our deliberations, and possibly even find a place on the Branch Council or its committees. They can then see what the funds are for, so that they can decide for themselves whether the expenditure is worth while. If they do not agree, they can propose reasonable alternatives.

In these days of materialism, when the profession is threatened by lay control, when powerful financial and gubernatorial powers seek to remove our autonomy, our only strength lies in a strong Association, representing all the many branches of the profession. No medical practitioner can do without the Association, and the Association is weakened by every practitioner who is not a member, or by those members who are merely obstructive in their outlook.

EXTERNAL CARDIAC MASSAGE

LOUIS MILNER, M.R.C.S., L.R.C.P., D.P.H., D.A.

Johannesburg Hospital and the University of the Witwatersrand

Even the lay press has given prominence to a 'Japanese method of resuscitation'[1,2] since the Editorial in this *Journal* on the efficacy of external cardiac resuscitation.[3] References from the professional journals are becoming too numerous to quote comprehensively, but one needs only to look at the correspondence columns of recent issues of the *Lancet* and the *British Medical Journal* to realize how acceptable the technique has become, and how many can now testify to its validity. Instances of so-called cardiac arrest have now received great attention and interest from the medical profession for so many years that many of us believe that its 'incidence' has greatly increased, and we forget that cardiac arrest is an inevitable concomitant of the process of dying. We now know that when cardiac arrest occurs from reversible causes, as it commonly does in operating theatres, there exists an alternative, or at least a preliminary, manoeuvre to the more exacting and dramatic method of direct manual cardiac massage.

The purpose of this paper is to emphasize the rôle of external cardiac resuscitation by reporting its successful use in two cases of clinical cardiac arrest which occurred during one week at the Johannesburg Hospital.

CASE REPORTS

Case 1

A European male, aged 61, was operated upon for internal fixation of a fractured femur. Anaesthesia was induced with 5% thiopentone (400 mg.) and maintained with 5 litres of oxygen and 2 litres of nitrous oxide per minute only. Although he was breathing well and without obstruction, his plethoric appearance deteriorated to the point of cyanosis, in spite of the fact that the oxygen percentage was increased to 50%. His pulse was thready, and just after the administration of pure oxygen was resorted to, all pulses disappeared and his colour became grey. Soon after this his respirations failed. While preparation for immediate thoracotomy was in progress, two firm thumps to the lower end of the sternum restored excellent pulses and, very soon after that, respiration. The patient gave no more reason for concern, except for some restlessness for about two hours postoperatively which may have been caused by the transient cerebral anoxia.

Case 2

A White baby of 8 months, weighing only 5¼ lb., was anaesthetized for oesophageal dilatation by Dr. L. Bonner. The infant had earlier undergone uneventful repair of a tracheo-bronchial fistula, but on this occasion went into sudden cardiac and respiratory failure after inhaling, for 5 minutes only, a mixture of nitrous oxide, oxygen, and halothane in about a 2% concentration. Firm thumps on the chest with the flexed lower limbs of the patient doubled up over the chest, as described by Rainer and Bullough,[4] restored the circulation, and the child made a complete recovery.

Neither patient had been intubated or given muscle relaxants.

SUMMARY AND CONCLUSIONS

Two patients with apparent cardiac arrest, treated successfully by external cardiac massage, are presented. In both there may have been merely circulatory insufficiency from ineffective, but still remediable, contractions, since the effect of intravenous noradrenaline as suggested by Churchill-Davidson[5] in similar circumstances was not evoked. Nevertheless, in patients in whom it is felt that the complete and characteristic picture of circulatory arrest is caused by cardiac arrest, it would appear thoroughly worth while to try external massage of the heart between the sternum and the vertebral column while the surgeon prepares for a direly urgent thoracotomy. Direct manual massage must, of course, remain the only certain way of initiating and maintaining an artificial circulation, at least until more convincing results, such as those of Kouwenhoven *et al.*[6] are available.

I wish to thank the Medical Superintendent of Johannesburg Hospital for permission to publish.

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ADDENDUM

Since submitting this article, I treated a third patient as described above:

A cachectic European male of 61 required a rib-resection and drainage of empyema. His blood pressure was 190/120 mm.Hg. He received 200 mg. of thiopentone followed by 30 mg. 'tubarine'. After endotracheal intubation he was moving in spite of being inflated with 50% nitrous oxide in oxygen, so 1% halothane was added to the mixture. After the patient was turned into the lateral position his blood pressure was 90 mm.Hg systolic, and the halothane was diminished to 0·5%. The skin incision revealed a 'bloodless' field, and at the same

time his pulses became unobtainable. Direct cardiac massage proved to be impossible owing to numerous adhesions, so the assistant surgeon performed closed-chest massage, that is, he compressed the heart between the lower end of the sternum and the vertebral column. Each time this was done I clearly felt the propagated impulse in the carotid arteries. Soon after, spontaneous beats reappeared, but ceased again after some minutes. Mephenteramine sulphate ('wyamine') was injected and external massage again yielded spontaneous beats after about half a minute. The subsequent course was uneventful and the patient recovered completely.

In the issue of the *Journal of the American Medical Association* dated 20 May 1961 there appeared several contributions about external cardiac massage in which it is shown that after roughly every 5 cardiac compressions the lungs require active, positive-pressure inflation. It is apparently not possible to rely on the cardiac massage movements to obtain pulmonary gaseous exchange; this has to be supplemented, if necessary by expired-air (mouth-to-mouth) respiration. No open thoracotomies have been performed for cardiac arrests for 2 years now at the Johns Hopkins Hospitals, and yet they have excellent results (with closed-chest resuscitation); a really impressive recovery rate, in fact, of 62%.

IN DIE VERBYGAAN : PASSING EVENTS

Dr. M. L. Simenhoff, of Cape Town, who is at present doing research work at the British Postgraduate Medical School, Hammersmith, has been appointed the Eli Lilly Medical Research Fellow (South Africa) for 1961.

Dr. Simenhoff will assume the tenure of his award at Harvard University, in the Department of Medicine, where he will work under Prof. John P. Merrill, Director of the Cardio-Renal Section, Peter Bent Brigham Hospital, Boston, USA.

* * *

Health and Tuberculosis Conference, Ibadan, Nigeria. The Chest and Heart Association, with the cooperation of the Federal Government of Nigeria and by kind invitation of the University College, Ibadan, is sponsoring a Health and Tuberculosis Conference to be held at the University College, Ibadan, Nigeria, from 26 to 31 March 1962.

This is a completely new venture and the aim is to encourage all workers in the field of tuberculosis in Africa. This is the sixth in the series of Commonwealth Conferences, which have previously been held in London.

Among the speakers will be eminent chest physicians from Great Britain and all parts of the world. The programme will include lectures and panel discussions on: Tuberculosis the greatest tropical menace, Tuberculosis and leprosy, Community infection — cause and remedy, Drugs — their value and limitations, The techniques of community surveys, Non-pulmonary tuberculosis, and Travelling clinics and village hygiene.

The Conference fee is R6.30 (three guineas) and accommodation in the University hostels will be available at moderate charges. Applications for further details should be made to the Conference Secretary, The Chest and Heart Association, Tavistock House North, Tavistock Square, London, W.C.1. and *not* to the University College, Ibadan, Nigeria.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 14 August at 5.10 p.m. in the Institute Lecture Theatre. Dr. D. J. Goldstein will speak on 'Some aspects of differential staining with basic dyes'.

* * *

Dr. Leon Goldman, of Cape Town, has left for Germany, Austria, and Italy, where he intends visiting various clinics. He will be attending the Israeli Medical Congress before returning to South Africa in the middle of September.

Mr. I. Norwich, F.R.C.S., of Johannesburg, and part-time Head of the Department of Surgery at Edenvale Hospital, will be going overseas on 12 August to present a paper on 'A 10-year survey of stabs in the abdomen' at the Congress of the International Society of Surgeons to be held in Dublin in September. He will be accompanied by his wife.

* * *

South African Paediatric Association (M.A.S.A.), Cape Town Sub-Group. The next meeting of this Sub-group will be held on Tuesday 8 August at 8.15 p.m. in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape. Dr. B. S. Jones will speak on 'Paralytic dislocation of the hip'. All those who are interested are invited to attend this meeting.

* * *

Second International Congress on Medical Librarianship, will be held in Washington, D.C. on 16 - 22 June 1963. The purpose of the Congress is to foster the development and improvement of medical library service throughout the world by bringing medical librarians together for the exchange of information on the problems, needs, and plans of medical libraries.

The Congress will provide free discussion of subjects relating to the improvement of education and training of medical librarians, the building of library resources, and international cooperation on bibliographic and other technical projects. The contribution of programme papers is invited, some of which will be selected for presentation at the Congress. Further information may be obtained from the Executive Secretary, Second International Congress on Medical Librarianship, c/o National Library of Medicine, Washington 25, D.C., USA.

* * *

Mr. Lee McGregor of Lister Building, Jeppe Street, Johannesburg, will retire from practice on 30 September and will leave for the Cape immediately. Colleagues who wish to have the records of patients should apply immediately since all records will be destroyed after the middle of September.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association will result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as those who change their addresses within the Union.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

CEPACOL

Mer-National Laboratories (Pty.) Ltd. announce the introduction of Cepacol, a new antibacterial lozenge and mouthwash/gargle, and supply the following information:

Composition. Cepacol contains Ceepryn (cetylpyridinium chloride), a new non-antibiotic antiseptic with a *deep* antibacterial action that penetrates the mucous barrier and kills a wide range of bacteria in 15 seconds. This bactericidal action lasts up to three hours after use. Among the bacteria destroyed by Cepacol are those commonly causing mouth and throat infections such as pharyngitis, tonsillitis, etc.

Cepacol is equally effective against fungus infections. Monilia albicans is killed on contact. In addition, Cepacol contains benzyl alcohol which has a powerful soothing action.

Cepacol tastes pleasant and leaves the mouth refreshed and clean — the gentle detergent action flushes out retained food particles and destroys odour-producing bacteria. Cepacol is virtually non-toxic and non-sensitizing — can be recommended even for young children.

Presentation. Cepacol lozenges: — Box of 24 (strip-packed). Cepacol mouthwash/gargle: Bottle of 8 fl. oz.

AMYLOZINE SPANSULE CAPSULES

SKF Laboratories (Pty.) Ltd. announce the introduction of Amylozine Spansule capsules, and supply the following information:

Composition. Trifluoperazine 2 mg. amylobarbitone 64·8 mg. (gr. 1).

Description. A preparation specifically developed for the treatment of agitation, apprehension and insomnia, Amylozine combines the highly efficient tranquillizing properties of trifluoperazine with the sedative action of amylobarbitone. Each component complements the other to provide a therapeutic action ideally suited for the symptomatic treatment of the chronic agitated psychoneurotic or acutely apprehensive patient. The onset of effect of Amylozine is prompt and, because the preparation is presented as a Spansule sustained-release capsule, is maintained for 12 hours with a single dose.

Indications. Treatment of chronic, agitated psychoneurotics and acutely apprehensive patients, whose state of mind is marked by insomnia, fatigue, vacillation, moodiness and fear.

Contraindications. Idiosyncrasy to barbiturates. Use cautiously in patients with cardiac or hepatic impairment.

Side-effects. Those associated with either component, e.g. dry mouth, dizziness, blurred vision, lassitude, or transient drowsiness may occasionally occur, but in the dosage recommended are minimal and infrequent.

Dosage. For adults and children over 12 years — one Amylozine spansule capsule morning and night as required, but not to exceed three capsules daily.

Presentation. Spansule capsules are available in containers of 30.

Further information may be obtained from SKF Laboratories (Pty.) Ltd., P.O. Box 38, Isando, Transvaal.

ACTIFED COMPOUND LINCTUS

Burroughs Wellcome & Co. (S.A.) Ltd. announce the introduction of Actifed Compound Linctus for the routine relief of coughs, and supply the following information:

Actifed Compound Linctus contains 1 mg. of triprolidine hydrochloride ('Actidil'), 20 mg. of pseudoephedrine hydrochloride, and 7·5 mg. of codeine phosphate and syrup base to 1 fluid drachm (3·5 c.c. approx.).

'Actidil' is the most potent and safe antihistamine. It promotes decongestion by its anti-allergic action. Pseudoephedrine, when given orally, has a marked vasoconstrictor effect which provides nasal and bronchial decongestion; it is also an effective bronchodilator. The combined effect is to promote ventilation of the respiratory tract, diminish 'post-nasal' drip, and allow drainage and expectoration of secretions. In addition, codeine phosphate acts as the antitussive in this combination to diminish 'useless' coughing.

Indications. For the relief of cough especially associated with congestion of mucous membranes of the respiratory tract in both adults and children. It is inadvisable for children under the age of 2 years.

Side-effects. Although pseudoephedrine causes virtually no pressor effect in patients with normal blood pressure, Actifed Compound Linctus should be used with caution in the presence of cardiovascular symptoms.

Presentation. Actifed Compound Linctus is available in 2-fl. oz. bottles and 20-fl. oz. bottles.

Dosage. For adults and children over 12 years of age: 1 to 2 teaspoonfuls every 4 - 6 hours. For children 2 - 12 years of age: ½ teaspoonful every 4 - 6 hours.

Further information may be obtained from Burroughs Wellcome & Co. (South Africa) Ltd., P.O. Box 10293, Johannesburg.

BOEKBESPREKINGS : BOOK REVIEWS

PNEUMOCONIOSIS

Pneumoconiosis.—Modern Trends. Report of meetings held in Birmingham (April 1959) and in Glasgow (January 1960). Pp. 144. Illustrated. R1.85. London: The Chest and Heart Assoc. 1961.

This symposium, being a report of papers read at meetings held in 1959 and 1960, is an excellent review of modern views on the pneumoconioses. Prominence is given to coal-pneumoconiosis, but silicosis and some newer pneumoconioses are also dealt with.

Two important points emerge: (1) that simple pneumoconiosis is not a disabling disease, but that complicated pneumoconiosis certainly is, and (2) that there is a large group of workers, in dusty trades, who suffer from bronchitis or emphysema, but who, because they show no radiological evidence of pneumoconiosis, are not eligible for compensation. Professor Gough summarizes modern views on this problem when he states that the problem is not so much the mechanism of the development of emphysema in pneumoconiosis, but whether emphysema developing in the absence of pneumoconiosis is related to occupation.

As a symposium, by a group of eminent workers, it is excellent, concise, and informative.

L.G.W.

BACTERIOLOGY

Bakteriologie und Serologie. Ausgewählte Untersuchungsmethoden für das bakteriologische und serologische Laboratorium (Dritte Auflage). von Dr. Lthar Hallmann, Pp XV+961. DM: 96—Stuttgart: Georg Thieme Verlag. 1961.

The third edition of this work exceeds the first one by 317 pages. It is in general a lavishly produced book, written by a leading expert for the bacteriological and serological routine practice. The work gives a comprehensive account of all the problems that may be encountered in connection with the running of a microbiological laboratory. Practising microbiologists and technologists will derive benefit from a study of this book, which they can safely rely on for guidance.

The arrangement of the subject matter is logical and each chapter is dealt with in such a manner as to produce a clear, concise and remarkably readable text. The book can be strongly recommended as an excellent overall summary in the field of Microbiology.

It would be to the advantage of English-speaking medical microbiologists and technicians to have this book translated into English.

H.D.B.

DERMATOLOGY

Atlas de Dermatologie en Couleurs (1952-61). De Graciansky, P. and Boulle, S. Paris: Maloine. 1961.

Works on dermatology, more than those on any other branch of medicine, require clear and accurate illustrations if they are to be intelligible to any but the initiated. Atlases of dermatology illustrated with aquatints and lithographs are curious and entertaining, but must have been more confusing than edifying to those who had to use them. Such atlases continued to appear long after the discovery of photography, but have declined in popularity, except with book-collectors, since the advent of colour photography.

Colour photographs would, theoretically, be essential for illustrating skin diseases, but there are drawbacks. Accuracy in colour reproduction does not always go with sharpness in outline, and few colour processes will always, or even often, illustrate the details of a dermatosis as well as the best black and white photographs. More important, a book on dermatology illustrated only with really good colour photographs is prohibitively expensive. The first problem has been solved in De Graciansky and Boulle's *Atlas de Dermatologie en Couleurs*. This work, finally completed after 9 years, contains 401 superb photographs that faithfully reproduce the colour changes of the dermatoses while giving sharpness of detail as good as can be obtained in black and white. All the common skin diseases and some of the rarities are depicted, and there are 1,300 pages of text with photomicrographs in black and white.

The authors must be congratulated on producing an atlas that is almost comparable to a visit to their seat of operations, the Saint-Louis Hospital in Paris. Unhappily, at 750 new francs, it is likely to be beyond the means of many who should possess it.

J. M.

BRIEWERUBRIEK : CORRESPONDENCE

CONVALESCENT HOME

To the Editor: We shall be glad if you would kindly remind your readers that the South African Red Cross Society's Convalescent Home, Raiders' Rest, in Avenue de l'Hermite, Sea Point, will take convalescent patients who do not require specialist nursing, for periods of convalescence not exceeding three weeks. This is of special value to patients who are not well off. No charge is made, but donations are accepted from patients who feel able to make some contribution towards their keep.

Applicants must be recommended by their doctor. Forms of application are available in Raiders' Rest itself or from the undersigned.

A. D. Forsyth Thompson
Regional Secretary

Red Cross House
14 Riebeeck Street
Cape Town
17 July 1961

SIMULTANEOUS INTRA-UTERINE AND EXTRA-UTERINE PREGNANCY

To the Editor: Following is the case report of a patient with simultaneous intra-uterine and extra-uterine pregnancy:

A young Mosuto woman was admitted to Quthing Hospital, Basutoland, at 9 a.m. on 27 May 1960. She had given birth to a small infant on 25 February 1960. The delivery was normal, but the child died a few hours later. Following this, however, she had persistent abdominal pain and episodes of fever. The abdomen did not completely regain its normal size.

Condition on admission. The patient was in extremis, grossly anaemic, and emaciated. Her temperature was 99·4°F., pulse 110 and her respiration 26 per minute. The abdomen was about the size of a 36-week pregnancy. The umbilicus was everted and the surrounding skin shiny and slightly oedematous. The abdomen was tender, dull to percussion, and fluctuant. No vaginal examination was made at this stage in view of the patient's poor condition.

Treatment. The umbilicus was incised without anaesthetic and 4,260 c.c. (nearly one gallon) of foul-smelling, thick, creamy, coloured pus removed. This contained strands of hair. Subsequently, cultivation of the pus yielded a mixed growth of non-haemolytic streptococci and Friedlander's bacilli, both of which were sensitive to 'chloromycetin'. The provisional diagnosis of an infected retained foetus was made and the patient given chloromycetin and penicillin. She was transfused with two pints of blood and her condition improved sufficiently for a laparotomy to be carried out on 3 June 1960. Under general anaesthesia with open ether the abdomen was opened by a midline sub-umbilical incision. This revealed a large abscess cavity in the depths of which could be palpated a macerated foetal face. This was surrounded by adhesions and was firmly fixed. Only small pieces could be removed digitally. As the cavity contained loops of bowel and since the patient's condition was still poor, no further attempt at removal of the foetus was made. The cavity was irrigated with normal saline, and penicillin powder was inserted. A large drain was left in each inguinal region and the incision was closed. Postoperatively a faecal fistula developed, but this closed spontaneously by 25 June 1960. Apart from this the patient made good progress and was discharged much improved on 2 August 1960.

Follow-up. The patient was next seen in October 1960. Her only complaint was of episodes of colicky abdominal pain. On examination her general condition was very good. The abdomen had regained its normal size, but a large indefinite mass was palpable in the lower abdomen. The bowel sounds were accentuated in this area and it was thought that the patient had a minor degree of intestinal obstruction from adhesions. Vaginal examination showed the uterus not to be enlarged and the mass was not palpable. The patient was not anxious to have further operative treatment and when last heard of had returned in good health to her husband in the Republic of South Africa.

The final diagnosis was that of a simultaneous intra-uterine and extra-uterine pregnancy, the former delivering three months previously, and the latter becoming macerated with subsequent abscess formation. The other interesting feature is the very large amount of pus evacuated, as opposed to ascitic fluid, which is often obtained in large amounts.

The above case was not completely investigated, but at the time there was no pathological laboratory and no X-ray department at the hospital.

Comment. The first case of simultaneous intra-uterine and extra-uterine pregnancy was described by Duverney in 1708, and this was discovered at postmortem examination. Since then over 400 cases have been reported in the literature. Several variations of degeneration may occur in the ectopic pregnancy, such as lithopaedion formation, mummification, maceration, and abscess formation. The above case is an example of the last. Cases have been described where the abdominal mass of the retained extra-uterine foetus has undergone gradual shrinkage noted from nine weeks to one year. An abdominal abscess has developed two years after delivery with discharge of faeces and foetal parts through the abdominal wall. Severe emaciation is often associated with the retention of a dead extra-uterine foetus.

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Registrar

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MEDEVES ASSOCIATION OF VEREENIGING

To the Editor: The Medeves Association of Vereeniging and neighbouring towns was started in October 1960 by Mrs. Y. Erasmus, wife of Dr. J. Erasmus. It originated because it was felt that a Christmas party should be provided for the children of doctors and dentists. The meetings held to discuss the Christmas party idea were pleasant, social occasions, and the idea grew to arrange these meetings regularly. As there are only four veterinary surgeons in this area, their wives were invited to join the group. Our group is probably unique in that it incorporates the wives of medical, dental and veterinary doctors — hence the name: Medeves.

Meetings are held once a month, each time at the home of one of the members, when 3 or 4 act as co-hostesses. Sometimes entertainment is provided, but often the meeting is simply a social 'get-together'.

We do not aim to do any fund-raising, except for the Benevolent Fund, and to this end a film show was organized in April 1961. The superintendent of the Vereeniging Hospital, Dr. P. Swanepoel, who has an international reputation for his film work, showed films, and an attendance of over 200—consisting chiefly of doctors and their families, raised £50 by donation. Part of this money was sent to the Dental Benevolent Fund and the rest to the Medical Benevolent Fund.

At our first meeting in October there were 12 wives. At the Christmas party there were over 60 children and at present over 70 notices are sent out each month. We are proud of our progress and think we are helping to bring the doctors (all 3 types) into closer contact and understanding with each other.

The present committee consists of: Chairlady, Mrs. Y. Erasmus; secretary, Mrs. E. Blake; treasurer, Mrs. M. de Beer; and other members, Mrs. van der Walt, Mrs. Berman (Meyerton), and Mrs. Backeberg (Sasolburg).

E. S. Adderley
On behalf of the Medeves Association

Vereeniging
Vaal River Branch (M.A.S.A.)
20 July 1961